A Dissertation on

"A RANDOMIZED CLINICAL TRIAL COMPARING PLAIN BUPIVACAINE AND DEXMEDETOMIDINE AS ADJUVANT TO BUPIVACAINE IN PAEDIATRIC CAUDAL ANESTHESIA"

Submitted to the

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfilment of the requirements

For the award of degree of

M.D. (Branch-X)

ANAESTHESIOLOGY

GOVERNMENT STANLEY MEDICAL

COLLEGE & HOSPITAL

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY,

CHENNAI, TAMILNADU

APRIL 2013

DECLARATION

I, **Dr .SUREKHA G** , solemnly declare that the dissertation, titled

"A RANDOMIZED CLINICAL TRIAL COMPARING PLAIN BUPIVACAINE AND DEXMEDETOMIDINE AS ADJUVANT TO BUPIVACAINE PAEDIATRIC CAUDAL ANESTHESIA"

, is a bonafide work done by me during the period of November 2011 to August 2012 at Government Stanley Medical College and Hospital, under the expert supervision of **Dr.P. CHANDRASHEKAR, M.D, D.A.** Professor and Head of Department Of Anaesthesiology, Government Stanley Medical College,
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This thesis is submitted to The Tamil Nadu Dr .M.G.R. Medical University in partial fulfilment of the rules and regulations for the M.D. degree examinations in Anaesthesiology to be held in April 2013.

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Date:

CERTIFICATE

This is to certify that this dissertation entitled **"A RANDOMIZED CLINICAL TRIAL COMPARING PLAIN BUPIVACAINE AND DEXMEDETOMIDINE AS ADJUVANT TO BUPIVACAINE IN PAEDIATRIC CAUDAL ANAESTHESIA",** Submitted by **DR.SUREKHA.G** to the faculty of ANAESTHESIOLOGY, The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the requirement in the award of degree of M.D. Degree, Branch - X (ANAESTHESIOLOGY), for the April 2013 examination is a bonafide research work carried out by him during the period of November 2011 to May 2012 at Government Stanley Medical College and Hospital, Chennai under our direct supervision and guidance of **Dr. KRISHNAN N**,chief and Professor, Department of Anaesthesiology at Stanley Medical College, Chennai.

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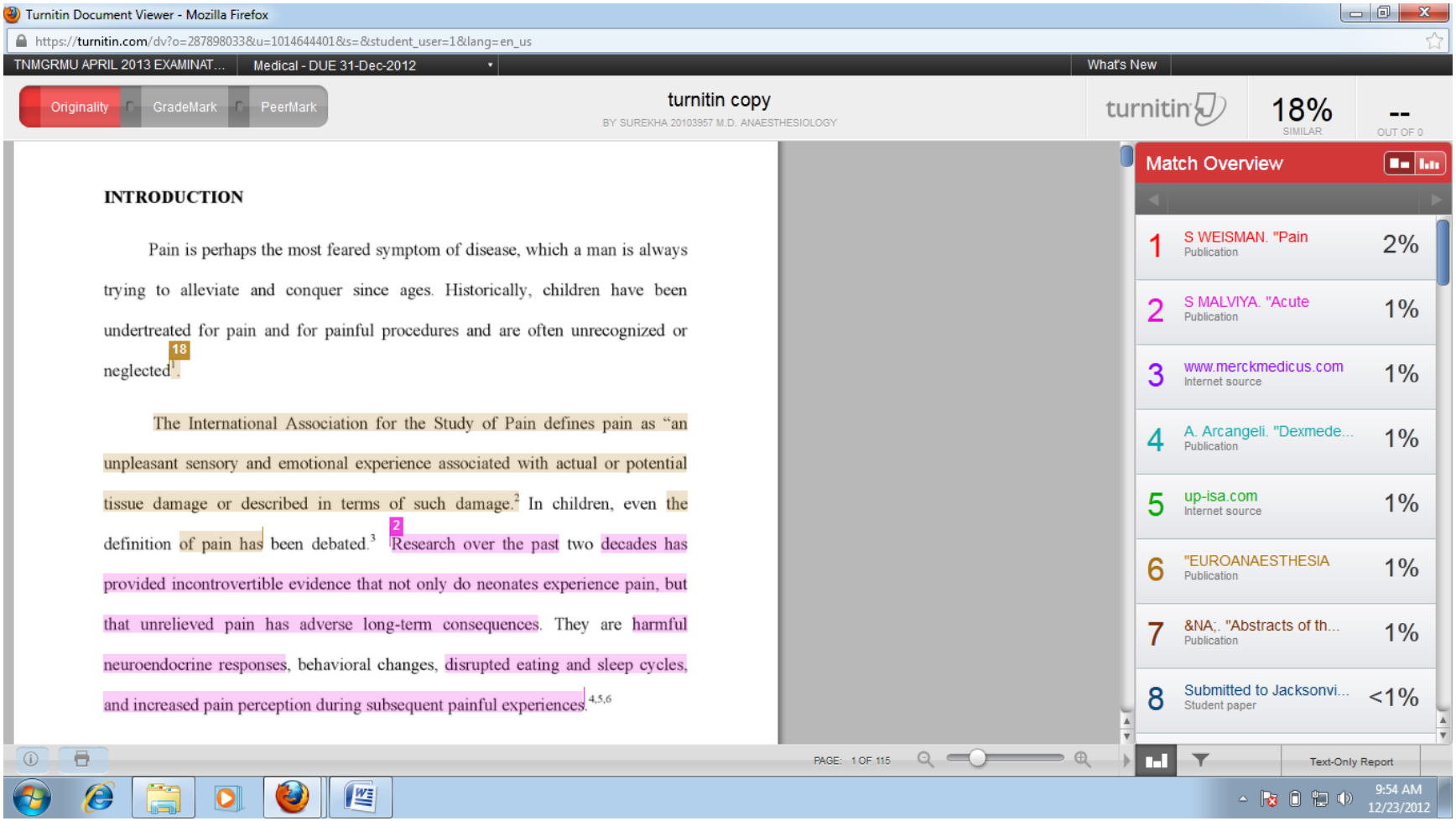
CONTENTS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INTRODUCTION
Pain is perhaps the most feared symptom of disease, which a man is always trying to alleviate and conquer since ages. Historically, children have been undertreated for pain and for painful procedures and are often unrecognized or neglected[1].
The International Association for the Study of Pain defines pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage.[2] In children, even the definition of pain has been debated.[3] Research over the past two decades has provided incontrovertible evidence that not only do neonates experience pain, but that unrelieved pain has adverse long-term consequences. They are harmful neuroendocrine responses, behavioral changes, disrupted eating and sleep cycles, and increased pain perception during subsequent painful experiences.[4,5,6]
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நோயாளி தகவல் தாள்

குழந்தைகளுக்கு முதுகு தண்டு மூலம் ஊசி செலுத்தி Dexmedetomidine as adjuvant with Caudal Bupivacaine அதன் வலி நிவாரணத் தன்மையை ஒப்பிடும் ஆய்வு

நோயாளிக்கான தகவல்கள் :

ஆராய்ச்சின் நோக்கமும், ஆதாரங்களும் :

உங்கள் குழந்தையை ஈடுபடுத்த திட்டமிடப்பட்டுள்ள இந்த மருத்துவ ஆராய்ச்சி ஆய்வானது, குழந்தைகளுக்கான அறுவை சிகிச்சையின் போது முதுகு தண்டுவடத்தில் ஊசி மூலம் மருந்து செலுத்தி அதன் வலி நிவாரணத் தன்மையை ஆய்வு செய்வதாகும்.

ஆய்வு முறை :

இந்த ஆய்வில் உங்கள் குழந்தை அறுவை சிகிச்சைக்கு செல்லும் முன் தூக்க மருந்து கொடுத்து அறைக்கு எடுத்து செல்லப்படும். அங்கு இரத்த நாளத்தில் சிறு ஊசி மூலம் மயக்க மருந்து கொடுக்கப்படும். பின்பு முதுகு தண்டுவடத்தில் வலி நிவாரண மருந்து ஊசி மூலம் செலுத்தப்படும். பிறகு அறுவை சிகிச்சை செய்யப்படும்.

ஆய்வில் உங்கள் உரிமைகள் :

உங்கள் மருத்துவப் பதிவேடுகள் மிகவும் அந்தரங்கமாக வைத்துக் கொள்ளப்படும். இந்த ஆய்வின் முடிவுகள் அறிவியல் பத்திரிக்கைகளில் பிரசுரிக்கப்படலாம். ஆனால், பெயரை வெளியிடுவது மூலம் உங்கள் குழந்தை அடையாளம் காட்டப்படமாட்டார்கள். இந்த ஆய்வில் உங்கள் குழந்தையின் பங்கேற்பு தன்னிச்சையானது மற்றும் காரணங்கள் எதையும் கூறாமலேயே நீங்கள் இந்த ஆய்விலிருந்து எந்த ஒரு நேரத்திலும் விலகிக் கொள்ளலாம். எப்படியிருந்தாலும் உங்கள் குழந்தைக்கு தகுந்த மயக்க மருந்து கொடுத்து அறுவை சிகிச்சை செய்யப்படும். இந்த ஆய்வில் ஏதேனும் பக்க விளைவுகள் ஏற்பட்டால் உங்கள் குழந்தைக்கு முழு சிகிச்சை மருத்துவ குழுவினரால் அளிக்கப்படும்.

நாள் :

பெற்றோர் கையொப்பம் /
இடது பெருவிரல் ரேகை
(மருத்துவரால் படித்துக்காட்டப்பட்டது)

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சிகிச்சைக்கு பின் இந்த மருந்தின் வலி நிவாரணத் தன்மை எவ்வளவு நேரம் இருக்கும் என்பதற்கும் மற்றும், பக்க விளைவுகளும் ஆராயப்படும்.

INTRODUCTION

Pain is perhaps the most feared symptom of disease, which a man is always trying to alleviate and conquer since ages. Historically, children have been undertreated for pain and for painful procedures and are often unrecognized or neglected[1].

The International Association for the Study of Pain defines pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage.[2] In children, even the definition of pain has been debated.[3] Research over the past two decades has provided incontrovertible evidence that not only do neonates experience pain, but that unrelieved pain has adverse long-term consequences. They are harmful neuroendocrine responses, behavioral changes, disrupted eating and sleep cycles, and increased pain perception during subsequent painful experiences.[4, 5, 6]

Till date, various methods and medications have been tried to provide post operative pain relief in pediatric population. Side effects of the pain medication have limited their use in children. For example, narcotics could cause respiratory depression, pruritis. Oral analgesics cannot be given during immediate post-operative period after general anesthesia due to the risk of vomiting and aspiration. Fear of needle stick in the case of parenteral analgesics poses problem in pediatric

population. Pain management is an integral part of practice of pediatric anesthesiologists[7].

Regional anesthesia in pediatric population is safe and effective. Along with providing post-operative analgesia, it reduces requirements of inhalational and intravenous agents with minimum sedation [7]. Caudal epidural anesthesia is the most commonly practiced regional technique in children for abdominal and lower limb surgeries.

Many local anesthetic drugs of variable concentration are used. Bupivacaine is a long-acting amide local anaesthetic that has provided reliable anaesthesia and analgesia with differential motor-sensory blockade for more than 40 years.[8, 9]

But the mean duration of surgical analgesia provided by long acting local anesthetic drug is only for 4-8 hrs during single shot caudal procedure. For this reason prolongation is achieved by addition of various adjuvants like opioids, clonidine, midazolam etc. Caudal opioid have advantages of prolonging duration of analgesia, but has side effects such as nausea, vomiting, pruritis and late respiratory depression[10].

Hence here is an attempt to study, addition of dexmedetomidine with striking lack of respiratory depressant effect, when given as adjuvant with caudal bupivacaine [8, 9.]

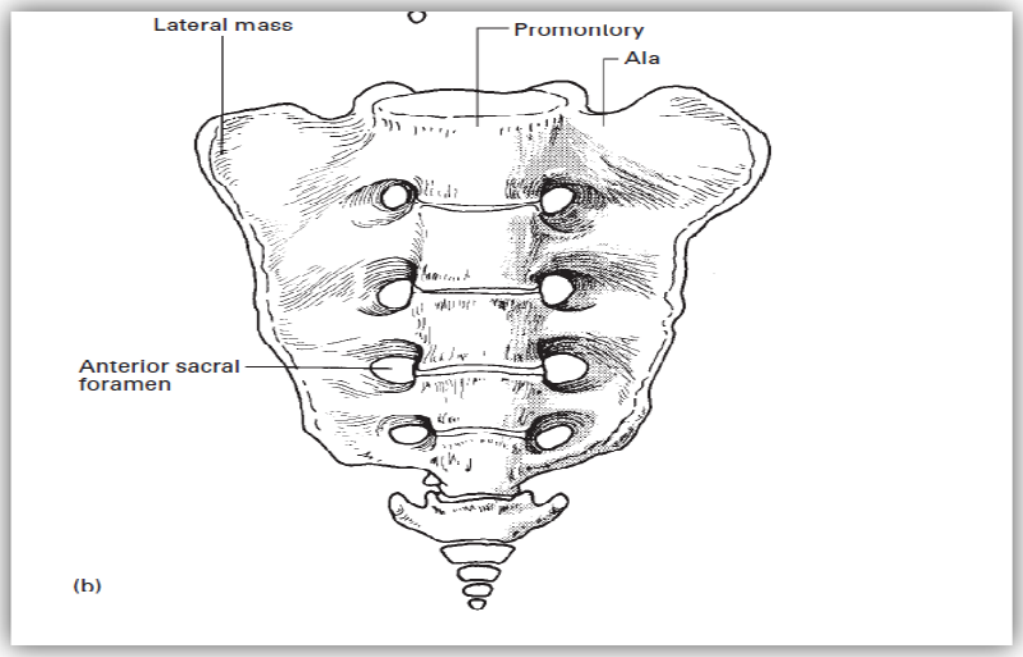
AIM OF THE STUDY

This study aims to compare plain bupivacaine and dexmedetomidine as adjuvant to bupivacaine in prolongation of post operative analgesia in pediatric caudal anesthesia.

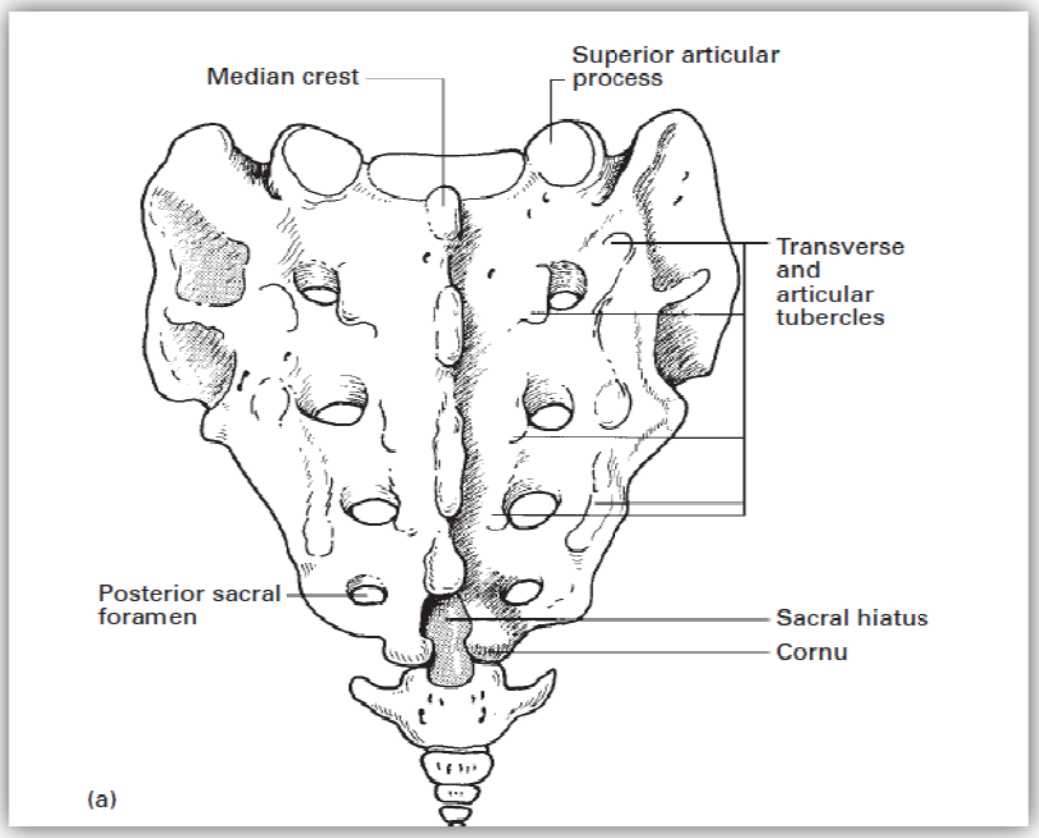
CAUDAL EPIDURAL ANALGESIA

ANATOMY OF SACRUM: [11, 12]

The Sacrum is triangular in shape, formed by the gradual fusion of the lamina of the five sacral vertebrae. It has concave anterior and convex posterior surfaces. Triangular shape of the sacrum is due to rapidly diminishing size of the lateral mass from above downwards. The apex below articulates with the Coccyx, Variations of this fusion are common and are responsible for the failure rate of caudal epidural analgesia. While the base has medial and lateral portions. The medial part represents the body of the 1st sacral vertebra and articulates with the corresponding surface of the body of the 5th lumbar vertebra. The lateral portions (alae) represent fused costal and transverse elements.



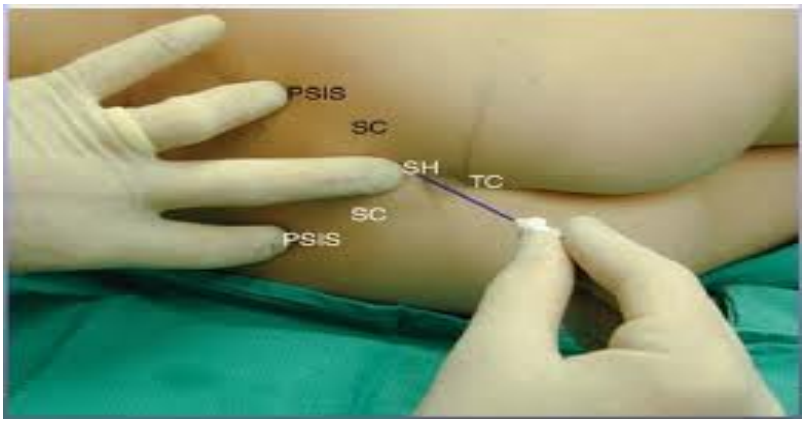
The anterior surface is concave and ridged representing fusion between the five sacral vertebrae. Lateral to the ridges are the four large anterior sacral foramina through which the anterior primary rami of the first four sacral nerves pass. These are formed due to the fusion of the transverse processes of the sacral embryologic segments. There are usually four such foramina, the fifth being absent. Local anaesthetic solution injected into the sacral epidural space pass freely through these foramina.



The posterior surface is rough and has greater interest for the anaesthetist. It is convex and in the midline runs a bony ridge, the median sacral crest with three or four rudimentary spinous processes. The fused vertebral arches forms the roof of the sacral canal

The Sacral hiatus is a deficiency of posterior wall resulting from failure of fusion of the lamina of the fifth sacral vertebra that communicates with the sacral portion of the vertebral canal. This hiatus is triangular in shape with its apex at the spine of fourth sacral vertebra.

In surface marking, it normally forms an approximately equilateral triangle with the two posterior superior iliac spines. There are bony prominences on the lateral margins of the space – the sacral cornua – which represent the inferior articular processes of the fifth sacral vertebra. The base of the hiatus is the superior surface of the coccyx. The posterior sacrococcygeal ligament, a continuation of the ligamentum flavum, is attached to the bony margin and covers the hiatus.



In some cases the apex of the hiatus is the third sacral spine, due to the absence of the third and fourth laminae, and occasionally the whole of the bony posterior wall is deficient. When the lamina of the fifth sacral vertebra is present, the hiatus may be very small with a diameter as narrow as 2mm making the introduction of a caudal needle almost impossible.

There are four pairs of posterior sacral foramina corresponding with the anterior ones. The sacral canal is triangular and is the continuation of the epidural space and the dural sac, which usually terminates at the lower border of the second sacral vertebra though occasionally it extends below this point. The caudal epidural space contains the sacral and coccygeal nerve roots and filum terminale and continuation of the epidural venous plexus. Fibrous bands may be present in the sacral epidural space dividing it into loculi which prevent the spread of local anesthetic solutions and may result in incomplete anaesthesia.

PHYSIOLOGICAL EFFECTS: [11]

Local anaesthetic administered into the epidural space blocks nerve conduction. Extent to which the drug causes nerve conduction block is determined by concentration and volume of the drug injected, sensitivity of different nerve fiber types and by the drug employed. Although all agents tend to block preganglionic B fibers more readily, followed by pain fibers, the order of blocked are touch, proprioception and motor fibers because of difference in the selectivity for different sensory fibers. However muscle relaxation is a usual feature, muscle tone being reduced by the loss of afferent side of the reflex arc even in the presence of good voluntary power.

Epidural administration of local anaesthetics produces regional effects, the distribution and extent of which are determined by the site and volume of injection. Local anaesthetics in epidural space act on the nerves as they traverse the epidural space or as they pass out through the intervertebral foramina into the paravertebral spaces or on the nerves in the subarachnoid space by diffusion through the dura. Among these the predominant site of action is in the region of the intervertebral foramina where the spinal nerves lose their protective dural sheaths.

Effects on Cardiovascular system:

The effects of caudal block on the cardiovascular system are minimal except in cases of high caudal block. The blood pressure falls are not significant, and they can be treated with vasopressor drugs. There is usually slowing of the pulse.

Effect on Respiratory system:

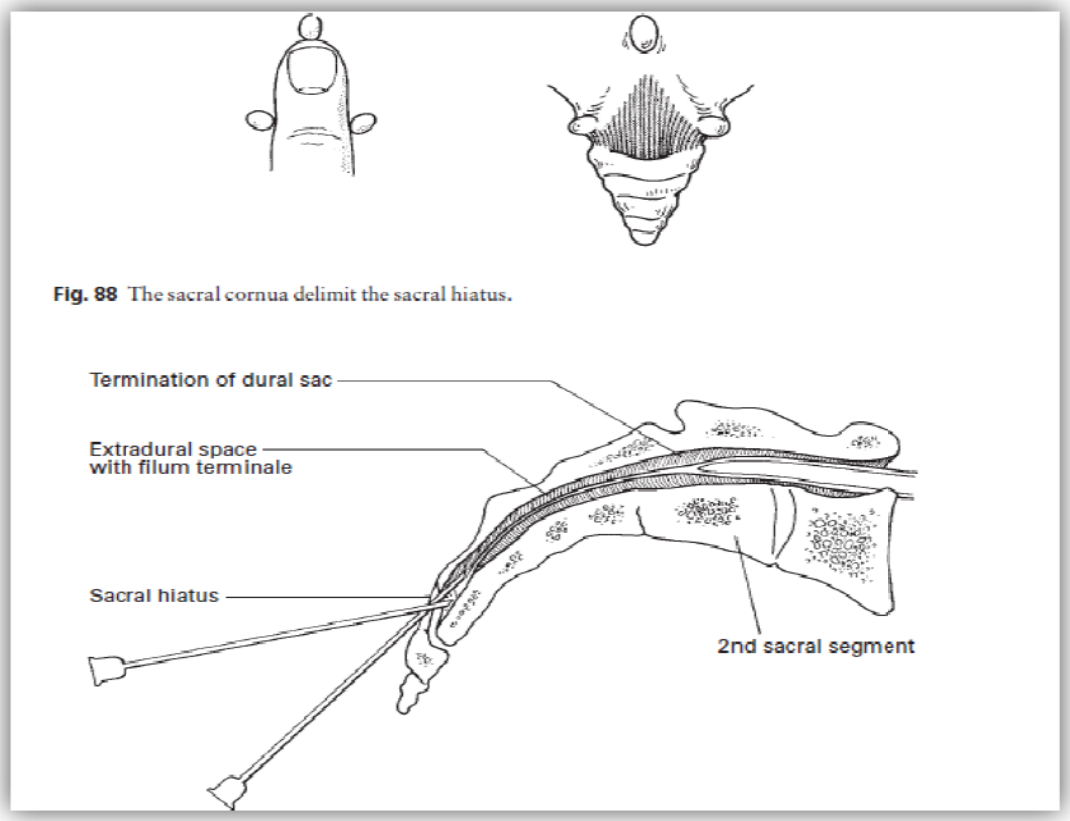
Respiration is usually not affected by caudal anaesthetization. When high caudal block is affected, there is little evidence of paralysis of intercostal muscles.

Effect on Gastrointestinal system: Caudal analgesia results in increased gastrointestinal tone that results in contracted bowel.

TECHNIQUE: [11]

The sacral hiatus at the lower end of sacrum is extremely easy to identify in infants and young children. The Sacral hiatus is relatively more cephalad in infants, thus the distance between the sacral hiatus and the end of dural sac is relatively short.

The lateral position is most often employed to perform a caudal block in children. To identify the sacral hiatus, we should first palpate the tip of the coccyx with the left index finger applying firm pressure to identify the coccyx and should move the finger gently from side to side proceeding in a cephalic direction.



The first double bony protruberances encountered are the two cornua of sacrum that define the sacral hiatus. The cornua should be marked either mentally or with the skin marking pen. The sacral hiatus can also be identified by drawing an equilateral triangle with the line joining the two posterior superior iliac spines forming the base and the sacral hiatus forming the apex.

After careful skin preparation, the sacral hiatus is again identified using firm pressure by the left index finger. Strict aseptic precautions should be maintained. A short beveled 23 gauge needle, preferably Crawford needle, is placed in the midline in the notch between the sacral cornua at an angle of about 45 with the skin (first position) and directed cranial, to penetrate the sacrococcygeal ligament, at which time contact is made with the anterior bony wall of the caudal canal(second position).

The needle then is depressed almost flush with the skin and then advanced into the sacral canal. The advancement should not be higher than a line joining the posterior superior iliac spines (S2 vertebra) since the dural sac ends between the first and second sacral vertebrae in majority of patients. Auscultation of sound over the caudal canal by injecting air (Oosh) or drug (Swoosh) can be done to confirm the presence of the needle in the caudal space. After negative aspiration for blood or cerebrospinal fluid, the appropriate amount of local anesthetic is injected and the child is placed in supine position.

CALCULATION OF VOLUME OF LOCAL ANAESTHETIC FOR CAUDAL ANAESTHESIA:

- **Armitage formula:**[13]

  Sacral dermatomes – 0.5ml/kg,

  Sacral and lumbar – 1ml/kg

  Midthoracic – 1.25ml/kg

- **Spiegel formula:**[14]

  For upper abdominal surgery, V= 4+ D-15/2

  Where V is the volume of local anesthetic in milliliters and D is the distance between C7 and the sacral hiatus in centimeters.

- **Satayoshi formula:**[15]

  V= D-13

  Where V is the volume of local anesthetic in milliliters and D is the distance from C7 to the sacral hiatus in centimeters.

- **Schulte-Scheinberg and Rahlfs formula:**[16]

  Volume in ml /spinal segment = 0.0558+ 0.09729(age in years)

- **Takasaki formula:**[17]

  Volume in ml / spinal segment = 0.056 (body weight in kg) - 0.002

PHYSIOLOGY OF PAIN

Pain is a complex constellation of unpleasant sensory, perceptual and emotional experiences and certain associated autonomic, psychological, emotional, and behavioral responses. Untreated pain in children, as the result of vaccinations and blood draws, surgery, headaches or repeated painful procedures, can have long-term effects.[18]

NEUROPHYSIOLOGY OF PAIN:[19]

A variety of chemical, thermal or mechanical insults can result in the sensation of pain. A mosaic of pain receptors or nociceptors in the body tissues ultimately project to pain centers in the brain. The somatosensory system is subserved by different groups of afferent fibers differentiated by their anatomy, rate of transmission, and sensory modality transmitted. The afferent fibers that relay pain information to the dorsal horn of the spinal cord and then on to the brain include small-diameter C-fibers and thinly myelinated A-delta fibers.

The dorsal horn if organized into fairly discrete lamellae. The primary afferent first-order synapses (nociceptive-specific neurons) are located in layers 1, 2 and 5 of the dorsal horn. Signals are then relayed rostrally to the thalamus and the cortex. In addition, afferent impulses are carried to the brainstem, limbic system, and hypothalamus to mediate many of the autonomic and affective

component responses to noxious stimuli. Deeper in the dorsal horn are located wide dynamic range neurons (WDR) that appear to be important in the development of hyperalgesia, or wind-up phenomenon. These neurons may be responsible for firing in pain syndromes that are not associated with obvious tissue-damage as well.

DEVELOPMENTAL NEUROBIOLOGY OF PAIN:

Nociceptive pathways in the periphery, spinal cord, and brain develop in a series of stages through the second and third trimester in humans. By 26 weeks` postconceptual age there is sufficient maturation of peripheral and spinal afferent transmission for the late-gestation fetus or preterm neonate to respond to tissue injury or inflammation with withdrawal reflexes, autonomic arousal and hormonal-metabolic stress responses. There are also changes in responsiveness after injury or repetitive stimulation indicative of central sensitization.[20]

It is important to understand that pain due to surgical procedures not only results in an immediate nociceptive response but also results in changes in the nociceptive activation pathways that lead to hypersensitivity, hyperalgesia and allodynia.[21, 22]

ASSESSMENT OF PAIN:

SELF-REPORTING TECHNIQUES:

- **Visual analog pain scale (VAS)**:[24]

It is often considered to be the gold standard for pain assessment. It is a 10cm horizontal line defined by "no pain" on the left end and "severe pain" on the right. It is used in older children and adolescents. The patient slides the cursor along the ruler until it reaches the level that represents the intensity of his pain. The other side of the ruler is graduated over 10mm and gives the investigator a numerical measure of pain. In children, the Verbal analog scale[30] (pain rated from 0(no pain) to 10 (most pain possible) may be more reliable.

- **Analogue Chromatic Continuous Scale (ACCS)**:[25]

The VAS has been modified for smaller children to equate pain intensity with colours in this scale. Instead of a line, the patient`s side of the scale is a wide band of colour ranging from pink for no pain to dark red for maximum pain, with increasing shades of red for intermediate degrees of pain.

- **Bieri – Modified:**[26,27]

This has Line drawings of faces from neutral to crying. This is mainly used for > 3 years children and has score from 0 -6 (original), 0 -5 or -10 (modified).

- **OUCHER Scale: (Beyer)**[28]

It is used in 3-12 years children. This up and down scale has photographs of a child in six increasing degrees of pain scored from 0 for the comfortable and calm face to 100 for the upset crying face.

BEHAVIOURAL AND COMPOSITE PAIN ASSESSMENT SCALES:

- **Premature Infant Pain Profile (PIPP)**:[29,30]

This is mainly used for Preterm and full-term neonates. Gestational age, behavioral state, heart rate, oxygen saturation, brow bulge, eye squeeze, nasolabial furrow are the indicators. It has 0 – 21 scoring.

- **Neonatal Infant Pain Scale (NIPS)**:[31]

This is based on facial expression, cry, breathing pattern, arms, legs, state of arousal. It is mainly used for Preterm and full-term infants. It has 0-10 score.

- **CRIES:**[32]

Crying, O2 saturation, Increased vital signs (Heart rate, Blood pressure), Expression and Sleeplessness are assessed in this scale. It is used in Full-term neonates and has a 0- 10 score.

- **FLACC**:[23]

This is mainly used in 2 months to 7 years children. Facial expression, Legs, Activity state, Crying, Consolability is the indicators used here. It has 0 -10 score. FLACC scoring provides simple framework to quantify pain in children who may not able to verbalize the presence or severity of pain. It is validated for the assessment of pain secondary to surgery, trauma, cancer, or other painful procedures for all preverbal children and children with mild to moderate cognitive impairment.

Interpretation FLACC behavioral scoring system:

Each category is scored 0-2 on the scale. Which results in a total score of 0-10

0 = relaxed and comfortable

1-3 = mild discomfort mild pain

4-6 = moderate discomfort or pain

7-10= sever pain or discomfort

- **Children`s Hospital of Eastern Ontario Pain Scale (CHEOPS)**:[33]

Cry, Facial expression, verbalization, torso position, touch (affected area); Legs are scored in this scale. It is used in 1-7 year age group. It has 4-13 scoring system.

- **COMFORT Score**:[34]

It is applicable for all ages. The indicators assessed are alertness, calmness/agitation, respiratory response, physical movements, heart rate, blood pressure, muscle tone, facial tension. It has 0 -40 score.

- **Hannallah Objective Pain Scale**: [35] .

It uses six parameters like Systolic blood pressure, Crying, Movement, Agitation, Posture and Verbalization of pain. It is scored from 0 – 12.

PHYSIOLOGICAL MEASURES:

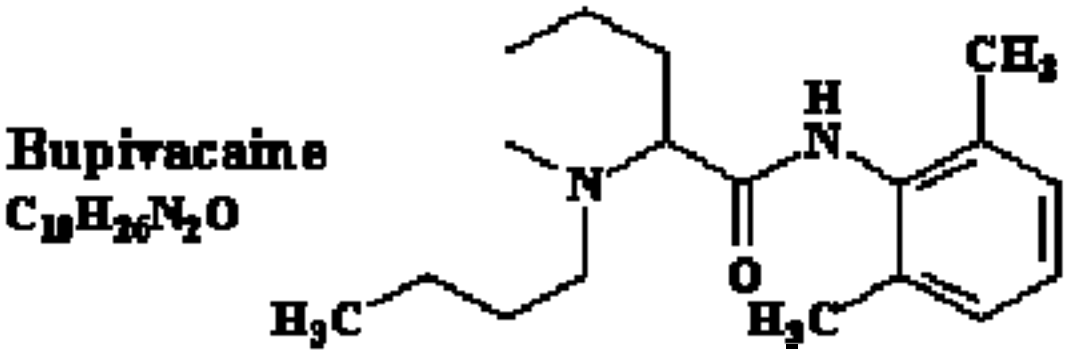
Observing changes in vital signs such as heart rate, blood pressure, respiration, oxygen saturation and sweating caused by pain removes the subjectivity of behavioral pain scoring methods, but these parameters may reflect changes for reasons other than pain and hence not often used.

PHARMACOLOGY OF BUPIVACAINE[36]

HISTORY

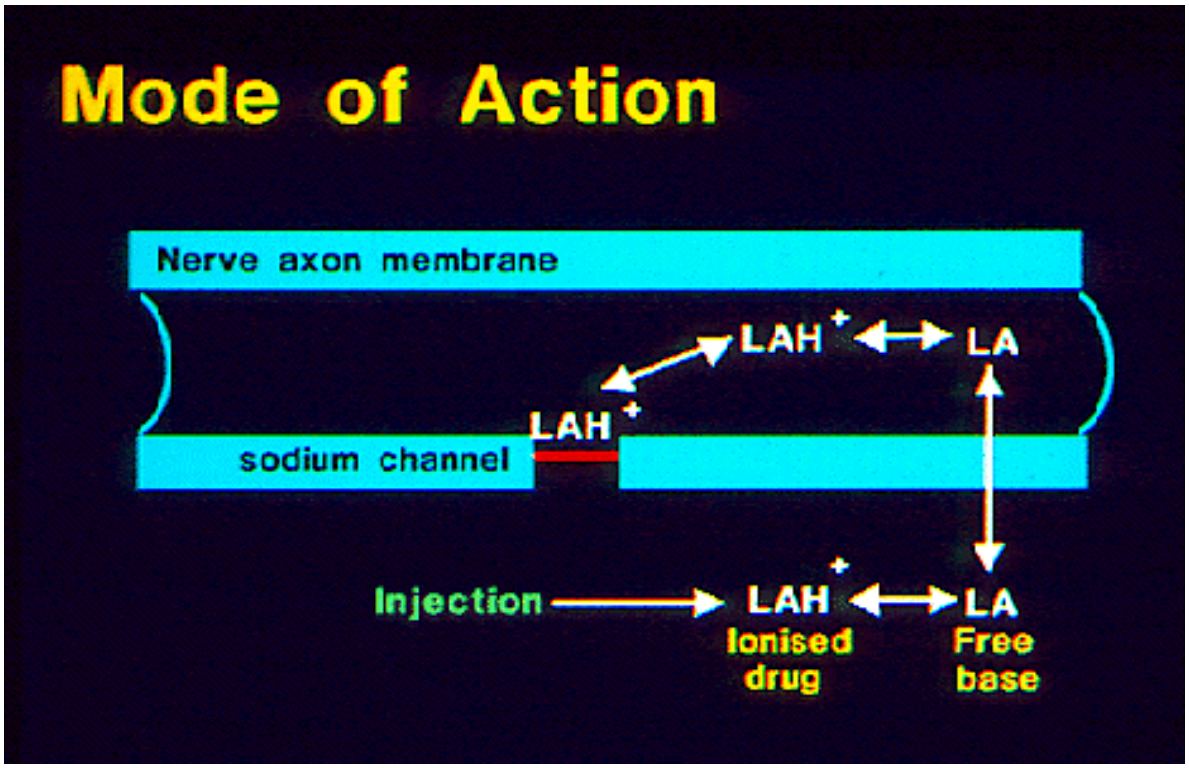
It is an amide linked local anesthetic synthesized by B.A.F. Ekenstam in 1957 and introduced into clinical practice by Talivuo in 1963.

CHEMICAL STRUCTURE: [36]



An amino amide local anesthetic having a benzene ring (lipophilic) at one end linked by an amide group to a tertiary amine (hydrophilic) on the other end of the molecule. It belongs to the group of pipecoloxylidide local anaethetics. All drugs in this group like mepivacaine, ropivacaine, levobupivacaine possess chirality due to the asymmetric carbon atom so that they may have optical isomers (enantiomers). The enantiomers may vary in their pharmacokinetics, pharmacodynamics and toxicity. Hence, administering a racemic drug mixture is, in reality, administration of two different drugs.[37] Bupivacaine is available as a racemic mixture with the S-enantiomer less toxic than the R form.

MECHANISM OF ACTION:



Local anaesthetics prevent transmission of nerve impulses (conduction blockade) by inhibiting passage of sodium ions through ion-selective sodium channels in nerve membranes.[36] they diffuse in their uncharged base form through neural sheaths and the axonal membrane to the internal surface of cell membrane sodium ion channels. They combine with hydrogen ions to form a cationic species which enters the internal opening of the sodium ion channel and binds with the channel in the inactivated-closed state. This produces blockade of sodium ion channel thereby decreasing sodium ion permeability and preventing depolarization of the cell membrane.

Binding affinities of local anaesthetics to the sodium ion channels are stereospecific thereby contributing to their differing potencies among the enantiomers. In addition to sodium ion channels, local anaesthetics block voltage-dependent potassium channels but with lower affinity. Other additional actions may include blockade of voltage-dependent calcium ion channels (L-type most sensitive) and their action on G-protein coupled receptors.[39]

Differential conduction blockade is illustrated by selective blockade of small C fibers and small- and medium-sized A fibers, with loss of pain and temperature and preservation of touch, proprioception and motor function at low concentrations of local anaesthetics.

PHARMACOKINETICS:[36]

The onset and duration of conduction blockade is related to the pKa, lipid solubility and extent of protein binding of the drug. A low pKa and high lipid solubility are associated with a long duration of action. Pka of bupivacaine is 8.1, lipid solubility is 95%.

ABSORPTION

The absorption of bupivacaine from its site of injection into the systemic circulation is influenced by the site of injection and dosage and use of epinephrine but the ultimate plasma concentration is determined by the rate of tissue distribution and the rate of clearance of the drug. Lipid solubility is important in the tissue redistribution as well as being a primary determinant of the drug potency with bupivacaine being highly lipid soluble and more potent. Protein binding will also influence its distribution and excretion that parallels the lipid solubility and is inversely related to its plasma concentration.

PHARMACOKINETICS

Elimination half life	210 min
Volume of distribution (Vdss)	73 L
Clearance (l/min)	0.47
Toxic plasma concentration	>3 micrograms/ml

Table 1

BIODEGRADATION AND ELIMINATION

Liver is the site of metabolism. Two major factors controlling the clearance of the amide linked local anesthetics are hepatic blood flow and hepatic function. The principal pathways are N-dealkylation, aromatic hydroxylation, amide hydrolysis and conjugation.

The mean total urinary excretion of bupivacaine and its dealkylation and hydroxylation metabolites account for >40% of the total anaesthetic dose. $Alpha_1$ acid glycoprotein is the most important plasma protein binding site of bupivacaine.

CLINICAL CHARACTERISTICS OF BUPIVACAINE[36]

Clinical use	Concentration	Onset	Duration (min)
Infiltration	0.25%	Fast	120-480
Nerve block	0.25-0.5%	Slow	240-960
Epidural	0.5-0.75%	Moderate	120-300
Spinal	0.5-0.75%	Fast	60-240

Table 2

Maximal dose 2mg/kg body weight

ADVERSE EFFECT AND COMPLICATIONS

Systemic toxicity:

This is due to an excess plasma concentration of the drug. Plasma concentrations are determined by the rate of drug entrance into the systemic circulation relative to their redistribution to inactive tissue sites and clearance by metabolism. The magnitude of the toxicity depends on dose administered, vascularity of the injection site, presence of epinephrine in the solution and the protein binding of bupivacaine.

Central Nervous System

Circumoral numbness is often an early symptom with restlessness, vertigo, tinnitus, and difficulty in focusing developing later. Further increases in the CNS concentration result in slurred speech and skeletal muscle twitching which signals the imminence of tonic-Clonic seizures. Seizures are classically followed by CNS depression, which may be accompanied by hypotension and apnea. The typical plasma concentration of bupivacaine associated with seizures is 4.5-5.5mic/ml. Hypoxia, Hypocarbia, hyperkalemia and acidosis can decrease the seizure threshold and increase CNS toxicity. The treatment includes oxygenation, ventilation and benzodiazepine or barbiturates help in termination of the seizures.

Cardiovascular system

The cardiovascular system is more resistant to the toxic effects of high plasma concentrations than is the CNS. Cardiac toxicity that results from high plasma concentrations occurs because; it blocks the inactivated state of the cardiac sodium and potassium (hKv1.5) channels[40]. The primary cardiac electrophysiologic effect of local anesthetics is a decrease in the rate of depolarization in the fast conducting tissues of Purkinje fibers and ventricular muscle.[40] Action potential and the effective refractory period are also decreased by local anesthetics.

Accidental intravenous injection of bupivacaine may result in precipitous hypotension, cardiac dysrythmias like premature ventricular contractions, Supraventricular tachycardia, atrioventricular heart block and Ventricular tachycardia that may be resistant to conventional resuscitative measures. Cardiotoxic plasma concentrations are 8-10 mic/ml.[41]

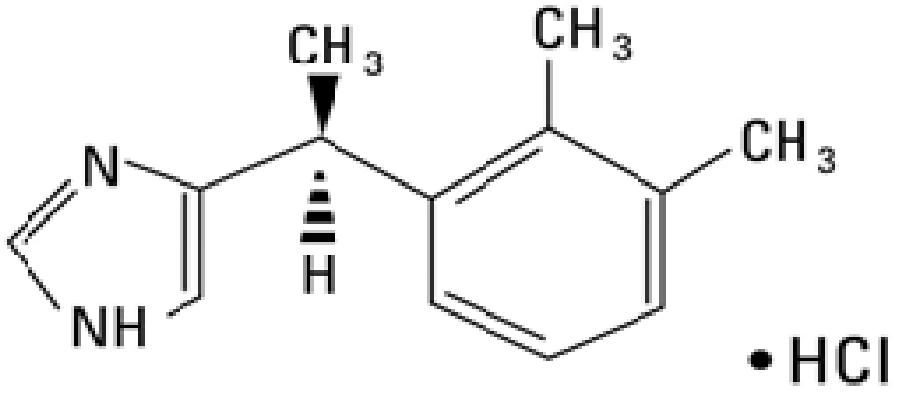
Moreover, bupivacaine depress the maximal depolarization rate of the cardiac action potential (Vmax) by virtue of its ability to inhibit sodium ion influx via sodium channels. This Vmax depression by bupivacaine is considerably more than lidocaine compared to ropivacaine that is intermediate between the two.[14] In addition, the rate of recovery from a use-dependent block is slower in bupivacaine-treated papillary muscles. Moreover, high blood levels of bupivacaine will prolong conduction time through various parts of the heart indicated by prolongation of PR interval and QRS complex. It also exerts dose-dependent negative inotropic action on cardiac muscle.

DEXMEDETOMIDINE:

HISTORY:

Dexmedetomidine is a potent, highly selective and specific alpha-2 adrenergic receptor agonist. It has both analgesic and sedative properties. The prototype of it, clonidine was initially developed in 1960s as a nasal decongestant for its vasoconstriction action. In 1966 it was established as a potent antihypertensive drug . Dexmedetomidine was approved in the USA in 1999 for sedation and analgesia in the intensive care unit. Since then it is used elaboratively in various other clinical conditions including anxiolysis, analgesia, as an adjuvant in both general anesthesia and central neuraxial blockade.

PHARMACOLOGY[42]: Structure: 4-(cs)-alpha, 2, 3-trimethyl benzyl imidazole mono hydrochloride.

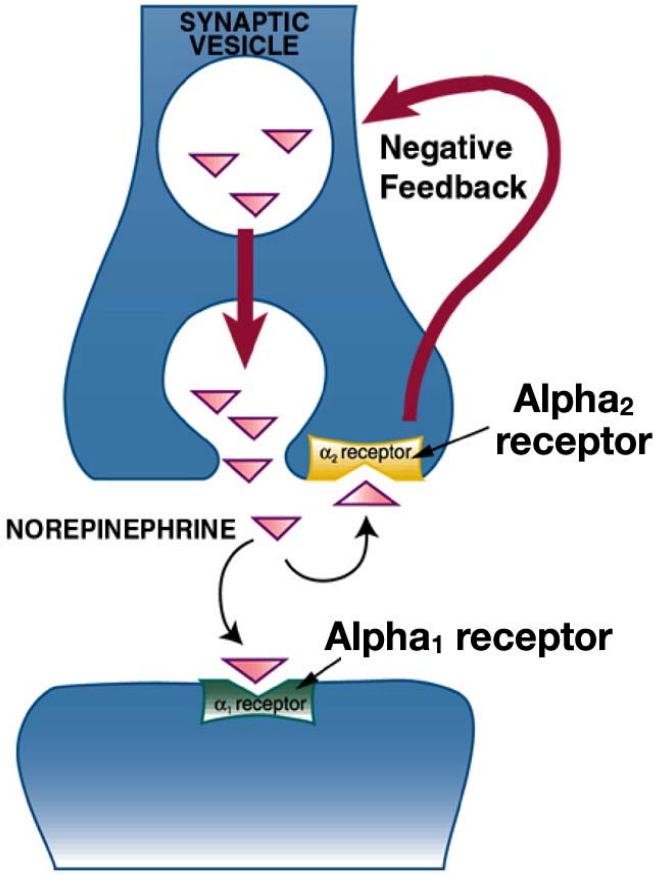


MECHANISM OF ACTION[42]:

Alpha-2 agonist acts at pre synaptic and postsynaptic receptors. The human alpha-2 receptors are classified into 3 subtypes, 2A, 2B and 2C.Drug binding at each receptor results in specific action.[4, 5]. 2A receptor agonist mediates sedative and anti nociceptive actions. Whereas 2B receptor activation causes vasoconstriction, resulting in hypertension at higher doses. 2C subtype modulates dopaminergic neurotransmission, hypothermia.

Dexmedetomidine can induce analgesia by acting at three sites: brain stem, spinal cord and in peripheral tissue. Dexmedetomidine is an selective alpha-2 agonist, with 2A subtype selectivity which makes it effective sedative and analgesic agent without undesirable cardiovascular effects from alpha-1 activation. In spinal cord alpha-2 receptors situated in the neurons of superficial dorsal horn especially lamina 2. Here pain transmission is reduced directly, by decreasing release of proprioceptive transmitter, substance P and glutamate from primary afferent terminals.

At cellular level drug binds to transmembrane receptor coupled to G protein and decreases adenylate cyclase and cAMP formation. Causes hyperpolarization via G-protein mediated potassium channel activation and decreases influx of calcium ions. Final result will be decreased action potential at spinal inter neurons and decrease in nor epinephrine release at locus coeruleus.



Pharmacokinetics

Following intravenous administration, dexmedetomidine exhibits the following pharmacokinetic parameters:

Distribution half-life (t1/2) 6 minutes;

Elimination half-life (t1/2) 2 hours;

Steady-state volume of distribution (Vss) 118 liters.

Clearance 39 L/h.

Distribution

The protein binding is 94%. Protein binding was similar in males and females. The plasma protein binding of dexmedetomidine is decreased in patients with liver disease.

The displacement of dexmedetomidine by other protein binding drugs like fentanyl, lignocaine, theofylline, ketorolac, and digoxin has been studied in vitro, and is found to be negligible.

Metabolism

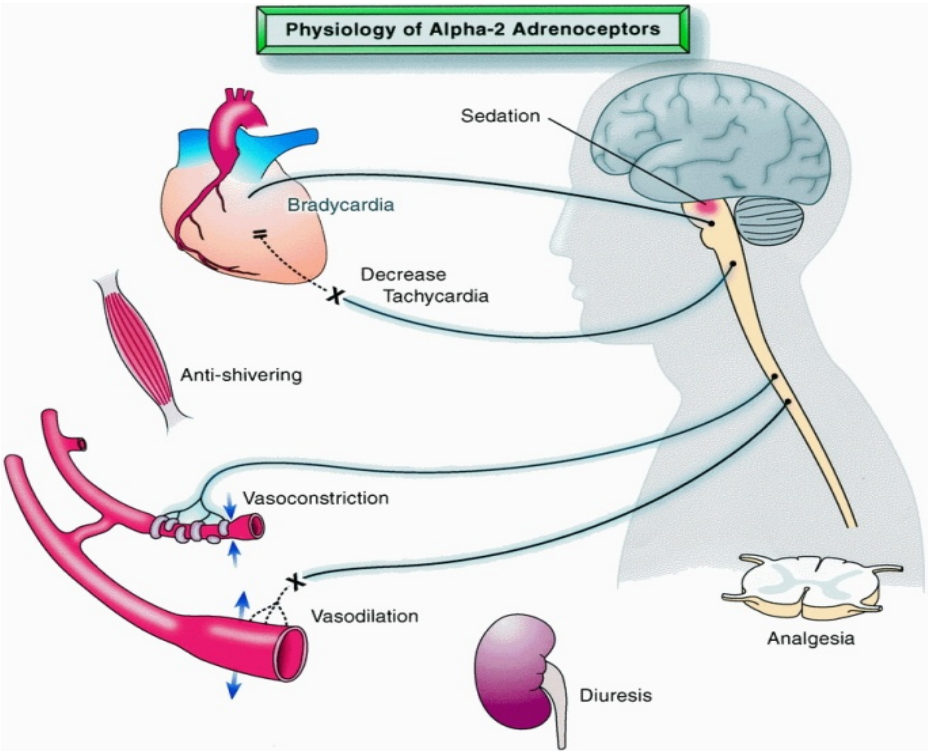
Dexmedetomidine undergoes complete biotransformation with very little unchanged form excreted in urine and feces. Biotransformation includes direct Glucuronidation to inactive metabolites and cytochrome P450 mediated metabolism.

N- glucoronidation to inactive metabolites, the glucoronide of 3-hydroxy- and 3-carboxy metabolite. N methylation of dexmedetomidine to generate 3-hydroxy N-methyldexmedetomidine, and dexmedetomidine-N-methyl O –glucuronide.

Elimination

After complete biotransformation 95% of the metabolites are excreted in urine and 4% in feces,

SYSTEMIC EFFECTS:



Effects on Cadriovascular system

The biphasic effect of dexmedetomidine on blood pressure is dependent on dose of the drug administered. It has both cental and peripheral action. At low doses decreases norepinephrine release at the nueroeffector junction and inhibits the neural transmission in symphethatic nerves[44] leading to decreased catecholamine levels in circulation. The final effect being slight fall in blood pressure and moderate fall in heart rate [45]. In patients with pre existing hypovolemia or vasoconstriction dexmedetomidine causes significant hypotension. Administered At higher doses, dexmedetomidine acts at alpha 2b receptors, producing hypertension

Respiratory system effects

Dexmedetomidine does not cause respiratory depression even when administered at higher doses. It was also demonstrated that combination of alpha$_2$ adrenoreceptor agonist opioids does not lead to further ventilator depression[47].

Central Nervous system effects

Dexmedetomidine action at alpha 2A receptors at locus ceruleous produces sedation. The mechanism is different from propofol and benzodiazepines, which acts at GABA receptors at cerebral cortex. It also has anxiolytic and analgesic properties[48].

Unique nature of sedation produced by dexmeditomidine is co-operative form. Patient will remain calm and readily arousable to verbal or tactile stimulus. This mimics natural sleep and preserves task performing ability and easy communication. When not stimulated patients will drift into sleep [49]. The drug has neuroprotective property by virtue of its action at 11 imidazolione imidozoline receptors in the brainstem and hippocampus[50]

Analgesia

Its action at Spinal cord is the major cause of analgesic action. By inhibiting release of substance P from the dorsal horn leads to primary analgesic effect.

Renal System Effects

Stimulation of alpha 2 receptors in the kidneys results in diuresis and natriurisis. It reduces efferent sympathetic outflow of the renal nerve and decreases the secretion of vasopressin. Dexmedetomidine antagonizes its effect on renal tubules. Also increases the release of atrial natriurisis peptide results in natriuresis[51]

Endocrine system effects

It Acts on symphethetic outflow and decreases circulating catecholamines levels. It attenuates stress response by inhibiting the secretion of adrenocorticotropic harmone (ACTH) and cortisol[51]. It directly inhibits insulin release leading to identifiable raise in serum glucose[52]

CLINICAL USES OF DEXMEDETOMIDINE:

1.Used as premedicant to decrease anxiety, and to alleviate intubation response when used at dose 0.5- 1mic/kg i.v or i.m.

2. as an adjuvant to general anesthesia drugs. Decreases over all requirement of narcotic and volatiles intra operatively.

3.Thoracic and cardiac surgeries: decrease in oxygen consumption, reduced occurrence of myocardial infarction. Cardioprotective in vascular surgeries

4. procedural sedation in MRI, endoscopy. Colonoscopy.

5. to facilitate awake intubation.

6. In obese patients posted for bariatric surgeries dexmed infusion decreased need of opioids.

7. In regional anaesthesia as adjuvant to local anaesthetics to prolong duration of analgesia.

8. In ICU to sedate ventilated patients.

ADVERSE EFFECT:

It causes dry mouth, bradycardia, hypotension, hypertension, atrial fibrillation, nausea. Mostly these side effects occur during the infusion of loading dose.

The effects of dexmedetomidine are reversible with antepimazole its use in humans not proven.

REVIEW OF LITERATURE:

El-Hennaway et al (-British Journal of Anaesthesia 2009)[53]Sixty patients (6 months to 6 yr) were evenly and randomly assigned into three groups in a double-blinded manner. After sevoflurane in oxygen anaesthesia ,each patient received a single caudal dose of bupivacaine 0.25% (1ml/kg) combined with either dexmedetomidine 2µg/kg in normal saline 1ml, clonidine 2µg/kg in normal saline 1ml or corresponding volume of normal saline. Addition of dexmedetomidine or clonidine to caudal bupivacaine significantly promoted analgesia time (16 hrs and 12 hrs respectively) than the use of bupivacaine alone (5hrs)[8].they found no significant difference in incidence of haemodynamic changes or side effects

Saadaway et-al. (**Acta Anaesthesiologica scandinavica. 2009 Feb**[52]**)** in a randomized double blind study involving 60 children aged 1-6 years undergoing sub umbilical operation, comparison was done between 1ml/kg of 0.25% bupivacaine and same dose of bupivacaine with dexmedetomidine 1µgm/kg. It was concluded that addition of dexmedetomidine to bupivacaine prolongs duration of postoperative analgesia (18.5 ± 2.8hrs versus 6.2±2.8hrs)[5]. Total consumption of rescue analgesic was significantly lower in Group BD compared with Group B ($P < 0.01$). There was no statistically significant difference in hemodynamics between both groups

Vijay G Anand, et al.(Indian Journal of Anaesthesia 2011)[54]

In a randomized, prospective, parallel group, double blinded study involving 60 children aged 6 months to 6 yrs for lower abdominal surgeries, comparison was done between 0.25% ropivacaine 1ml/kg with dexmedetomidine 2µgm/kg and the same dose of ropivacaine with 0.5 ml normal saline. It was concluded that addition of dexmedetomidine to ropivacaine prolongs duration of postoperative analgesia (14.5hrs versus 5.5 hrs)[6]

Mausumi Neogi et al-(J Anaesth Clin Pharmacol 2010)[55]

Authors studied effect of clonidine with ropivacaine, dexmedetomidine with ropivacaine, and plain ropivacaine in caudal anaesthesia in 75 children undergoing elective inguinal hernia repair surgeries. Patients received 1ml/kg of 0.25% ropivacaine caudally. Group C patients received 1ml/kg of 0.25% ropivacaine and 1µgm/kg clonidine. Patients of group D were given 1ml/kg of 0.25% ropivacaine and 1µgm/kg dexmedetomidine. The mean duration of analgesia was 6.32±0.46 hrs in group R, 13.17±0.68 hrs in group C and 15.26±0.86 hrs in group D. they concluded that the addition of alpha 2 agonists clonidine or dexmedetomidine prolongs post operative analgesia. Authors also observed that all the patients were hemodynamically stable during and after surgery.

K Sudheesh and SS Harsoor et al (IJA 2011)[56]

Authors discussed various uses and routes of administration of dexmedetomidine in clinical practice of anaesthesia in article they have mentioned that dexmedetomidine has been used in children through caudal route successfully at the doses 1to 2mcg/kg with local anesthetics bupivacaine and ropivacaine to prolong post operative analgesia without undue side effects like hypotension and bradycardia.

Xiang Q, Huang Zhao et al (British journal of anesthesia 2012)[57]**:**

Authors studied use of dexmedetomidine use in caudal anesthesia as an adjuvant with bupivacaine in attenuating response to hernia sac traction in terms of heart rate and rise in systolic blood pressure. Study was done in patient's age less than 3 years and more than 1 year undergoing unilateral herniotomy. They found that there was decreased response (3.33%) in group receiving 1mcg/kg dexmedetomidine compared to (43.33%) patients responding to tractional pain in plain bupivacaine 0.25% . they also noticed better duration of analgesia in dxmedetomidine group with decreased consumption of opioid-fentanyl post operatively.

Merkel et al: ((Peadiatric nursing 1997)[23]

Evaluated reliability and validity of the FLACC pain assessment tool in 89 children aged 2 months and 7 years who underwent variety of surgeries. They concluded that FLACC scoring provides simple framework to quantify pain in children who may not able to verbalize the presence or severity of pain

Sheta et al (international journal of pediatrics 2009):

Authors compared different doses of oral midazolam for premedication in paediatric age group. Doses compared were 0.5mg, 0.75mg/kg, and 1mg/kg midazolam. Observed for effectiveness in anxiolysis during parental separation and venepuncture. They found 0.5mg/kg oral midazolam as effective and acceptable dose as premedication and does not alter recovery time after general anaesthesia.

METHODOLOGY

STUDY DESIGN:

This was a Prospective Double blinded Randomized Comparative clinical trial conducted in Government Stanley Hospital, paediatric surgery department Chennai from April 2012 to August 2012. Children who were admitted to the paediatric surgery department for elective lower abdominal and perineal surgeries satisfying selection criteria were included in the study.

RANDOMIZATION:

The randomization was done by assistant, using simple lot system. We wrote equal number of letter A and B (50 envelopes contained letter A and 50 envelopes letter B) in a closed envolopes. Patients were asked to pick up one envelope randomly. Patients were assigned in a group whichever letter the envelope contained. The drug preparation was made by the assistant based on selected patients group. Caudal block was performed by the investigator, Intra operative monitoring and post operative observations were made by the same

BLINDING: The patient's parents/guardians were not aware, to which group the child belongs to. and the investigator was also blinded as the randomization and drug preparation was done by assistant who was not involved in the study.

DRUG PREPARATION:

Group A: 1ml/kg of 0.25% bupivacaine was prepared from 0.5% bupivacaine by adding equal volume of distilled water.

Group B: dexmedetomidine available as 100mcg/ml was diluted with 9ml of distilled water, into 10mcg/ml. Based on weight 2mcg/kg added to 0.5% bupivacaine and solution was made up to 1ml/kg of 0.25% concentration by adding distilled water.

Children who were included in the study, received 1ml/kg of study solution which was not labeled. The study blinding was broken, after the completion of all 100 cases and data sent for statistical analysis, Using observational data two groups were compared prospectively and statistic analysis made to derive conclusions.

SAMPLE SIZE CALCULATION:

Before the start of the study ,Pilot study was done with a sample size of 10 patients in each group, to decide on sample size. The mean and standard deviation of duration of post operative analgesia was calculated from pilot study. The sample size was calculated based on the formula given in NTI Bulletin 2006. (Sample size determination in health studies, V. K. Chadha,, National Institute Bulletin 2006,

42/3 & 4m 55-62.).From the pilot study got the value of mean and standard deviation.

Duration of post operative analgesia of Group-A (3.59 ± 0.59) and Group-B (15.14 ± 2.02).

$n = ([Z_{1-\alpha/2} + Z_{1-\beta}]^2 (2\sigma^2)) / (d)^2$ = (8.98 * 1.44) /0.13 = 99.5

Duration of Analgesia in a pilot study with 10 children in each group:

$Z_1 - \alpha/2$ = 1.96 (5%)

$Z_{1-\beta}$ = 1.037 (85 % Power)

$[Z_{1-\alpha/2} + Z_{1-\beta}]^2 = (1.96 + 1.037)^2 = 8.98$

$S = (s_1 + s_2) / 2$

$S = (0.69+0.98)/2 = 0.72$

$S^2 = (0.72)^2 = 0.52$

$2\sigma^2 = 0.52*2 = 1.44$

d = (Mean1 –Mean2)= (9.12-9.48)= 0.36

$d^2 = 0.13$

From the above calculation sample size was decided as 100

CRITERIA FOR PATIENT SELECTION:

After complete physical examination and basal investigations children were selected based on criteria:

2-7 years age

either male or female sex,

belonging to ASA I or II physical status,

Children undergoing elective lower abdominal or urologic surgeries like Herniotomy, Orchidopexy, appendicectomy, Procesus vaginalis sac ligation, Circumcision and Urethroplasty were included in the study.

EXCLUSION CRITERIA:

The children with the following problems were excluded from the study:

- Local infection in the Caudal region
- Preterm neonate
- Pre-existing Neuromuscular disease
- Congenital anomaly of the lower back
- Mental retardation, Delayed development
- Bleeding disorders or coagulopathy
- Parent refusal for the procedure.

METHODOLOGY:

After Obtaining clearance from the Institutional Ethics Committee of the Stanley Medical College, Chennai-1. The study was explained in detail to parents before including child in trial and written Informed Consent obtained from them.

The children were fasted for 6 hours for solids and 2 hours for clear liquids[37]. All children were premedicated with oral Midazolam syrup (2mg/ml) 0.5mg/kg45 minutes before surgery. EMLA patch was applied over dorsum of hand at the same time. They were brought into the operation theatre. The baseline parameters like heart rate, oxygen saturation; blood pressure measured.Precardial stethoscope was attached. intravenous access was secured with 22G intravenous canula . Intravenous fluid was started with Ringers lactate.

Inj. glycopyrolate 0.01mg/kg and inj fentanyl 1mic/kg i.v. administered. Oxygenated with 100% oxygen, Induced with inj thiopentone 5mg/kg. patients were intubated under direct laryngoscopy with appropriate size oral uncuffed endotracheal tube, 45 seconds after injecting 1mg/kg of inj suxamethonium. the tube position confirmed by auscultation and secured with elastoplaster. Anesthesia Maintained with 50% N20 and o2 50% mixture and sevoflurane 1%. Inj atracurium 0.1mg/kg given after recovery from suxamethonium and repeated when required.

CAUDAL BLOCK:

The children were placed in left lateral position after general anesthesia. Under strict aseptic precautions, patient's lower back was painted and draped. After palpating the landmarks and sacral hiatus, new 22G hypodermic needle was introduced in Caudal epidural space. To detect and avoid an inadvertent intravascular or subarachnoid injection, drug was injected after aspiration was negative for CSF or blood. Assistant who was not involved in the study, who prepared drug solution as above, handed over a unlabeled syringe for caudal. 1ml/kg of study solution was administered slowly, while monitoring vital signs and ECG. Then the patients were placed in Supine position, tube position reconfirmed. . The surgeons were requested to put the incision, 10 min after Caudal block, as the average onset time of caudal is around 10 min.

Heart rate, Blood pressure and Oxygen saturation was monitored continuously; values were recorded, after intubation, during caudal procedure and at the time of incision, then every 10 minutes interval till the end of surgery. If the patient responded to the surgical incision with a greater than 15% increase in Systolic Blood pressure or 15% rise in Heart rate ,it was planned to give Inj. Fentanyl 1 microgram/kg i.v.

Significant bradycardia requiring intervention was defined as fall in heart rate more than 20% from base line[38] and was planned to treat with injection Atropine 0.02 mg/kg. Significant hypotension requiring treatment was defined as more than 20% fall in Systolic blood pressure from baseline or systolic blood pressure less than 90 mmHg, and was planned to treat with intravenous fluid bolus 10ml/kg 0.9% normal saline.

At the end of the surgery, residual neuromuscular blockade was reversed with Inj. glycopyrolate 0.02mg/kg i.v. and with Inj. Neostigmine 0.05mg/kg i.v. The child was extubated when patient was hemodynamically stable, opens eyes to verbal commands and moves upper limbs purposefully. When the child was able to maintain room air saturation > 98% shifted to the recovery room for Observation.

POST-OPERATIVE PERIOD:

Post-operatively, patient was monitored closely in recovery room for 2 hours and later in post operative ward for 24 hours . Pulse rate, Systolic Blood Pressure and oxygen saturation recorded every 15min for first 2hrs and every one hour once for next 4 hours and then 6th hourly. Complications such as hypotension, bradycardia, postoperative nausea and vomiting (PONV), shivering were noted.

Apart from vitals, quality of Analgesia was assessed by using FLACC Objective Pain Scale[43] every 2nd hourly for first 12 hours, then 6th hourly.

Categories	Scoring		
	0	1	2
Face	Smile or no particular expression	Occasional grimace from, withdrawn, disinterested	Frequent to constant frown, clenched jaw, quivering chin
Legs	Normal position or relaxed	Uneasy, restless, tense	Kicking, or legs drawn up
Activity	Lying quietly, Normal Position, Moves easily	Squirming, shifting back and forth, tense	Arched, rigid, jerking
Cry	No cry (awake or asleep)	Moans or whimpers, Occasional complaint	Crying steadily, screams, sobs, frequent complaints
Consol ability	Content, relaxed	Re assured by occasional touching, hugging, or talking to, distractible	Difficult to console

Duration of analgesia was defined as "the time interval from the administration of caudal block and the requirement of first rescue analgesia for the patient". This time was noted. Inj. Fentanyl 1mcg/kg was given as rescue analgesia. Subsequent pain was treated with paracetamol rectal suppository 15mg/kg[70] when the FLACC pain score equals or exceeds 4.All analgesic requirements were noted in terms of number of times analgesic required in 24 hours. PONV treated as needed with ondansetron 0.06mg/kg. Bradycardia was planned to treat with injection Atropine 0.02mg/kg, Hypotension was planned to treat with 10ml/kg normal saline.

PARAMETERS OBSERVED:

1. Duration of analgesia.
2. Analgesia requirement in 24 hours.
3. FLACC score at regular intervals.
4. Hemodynamic changes in terms of heart rate and blood pressure.
5. Other side effects like nausea, vomiting and shivering.

STATISTICAL ANALYSIS

A sample size of 100 was decided based on pilot study results and Randomized into two groups consisting 50 children in group A and 50 in group B.

Data was expressed as mean+/- standard deviation. Quantitative variables like age sex and weight were analysed using student`s t-test. For continuous

variables like heart rate, blood pressure, duration of analgesia in hours, FLACC score, Chi-square test applied . When using the above statistical tests to compare the mean among the two groups, a p-value of less than 0.05 was taken as significant. All values were rounded off to a maximum of two decimals.

The children in each group were comparable in distribution in terms of age, weight, sex and basal parameters.

OBSERVATIONS AND RESULTS

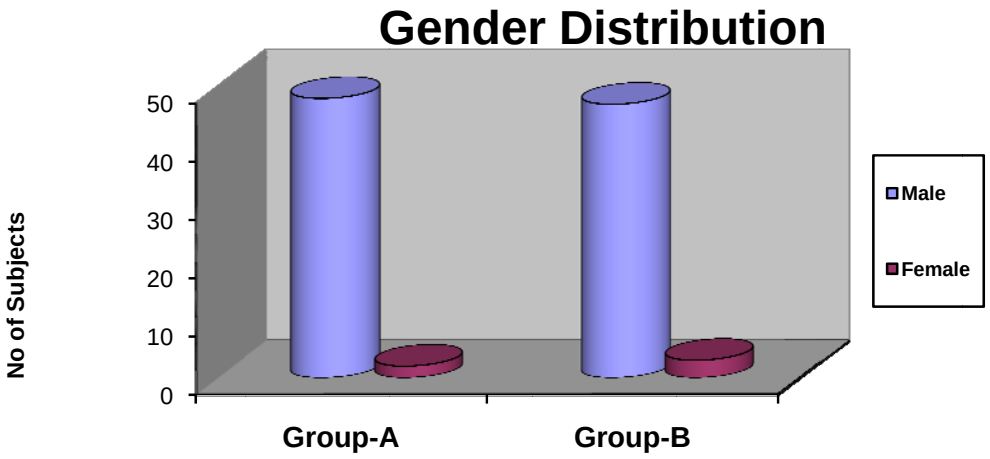
Demographic variables:

GENDER DISTRIBUTION:

There was no significant difference between two groups in terms of gender distribution. Among the 50 children in Group A, 48 were boys and 2 were girls whereas in Group B, 47 were boys and 3 were girls.

Table – 1 SEX distribution of the sample:

Sex	**Group-A N=50**		**Group-B N=50**		**Total N=100**	
	N	**%**	**N**	**%**	**N**	**%**
Male	48	96	47	94	95	95
Female	2	4	3	6	5	5
Chi-square value	**0.211**					
Df	**1**					
p value	**0.45 (Not Significant)**					

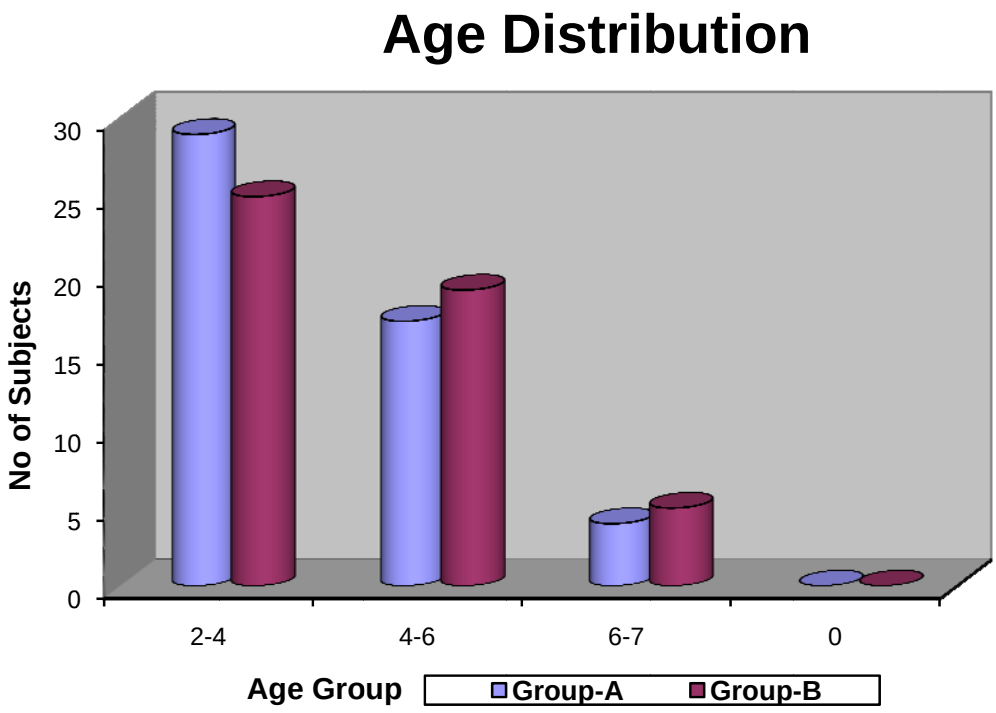


Age Distribution:

Both groups were comparable in terms of age, the mean age being similar around four and half years in both groups.

Table-2

	Group-A	Group-B
Mean	4.42	4.74
Sd	1.45	1.59
t-Value	**1.05**	
Df	**98**	
p-value	**0.30 (Not Significant)**	



WEIGHT DISTRIBUTION:

Weight was comparable between two groups.

The average weight in both the groups was around 12 kg with no significant difference.

Table-3

	Group-A	**Group-B**
Mean	11.72	11.86
Sd	2.19	2.32
t-Value	**0.31**	
Df	**98**	
p-value	**0.78 (Not Significant)**	

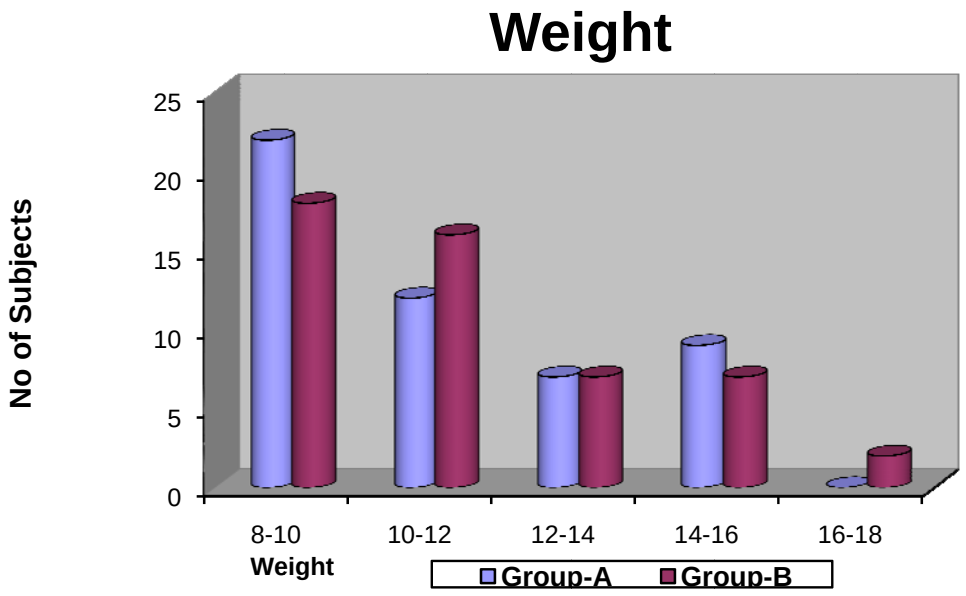


Table-4

ASA Status

	Group-A		Group-B	
	N	**%**	**N**	**%**
1	48	96	48	96
2	2	4	2	4
Total	**50**	**100**	**50**	**100**
Chi square Value *	**0**			
Df	**1**			
Significant	**1.000 (Not Significant)**			

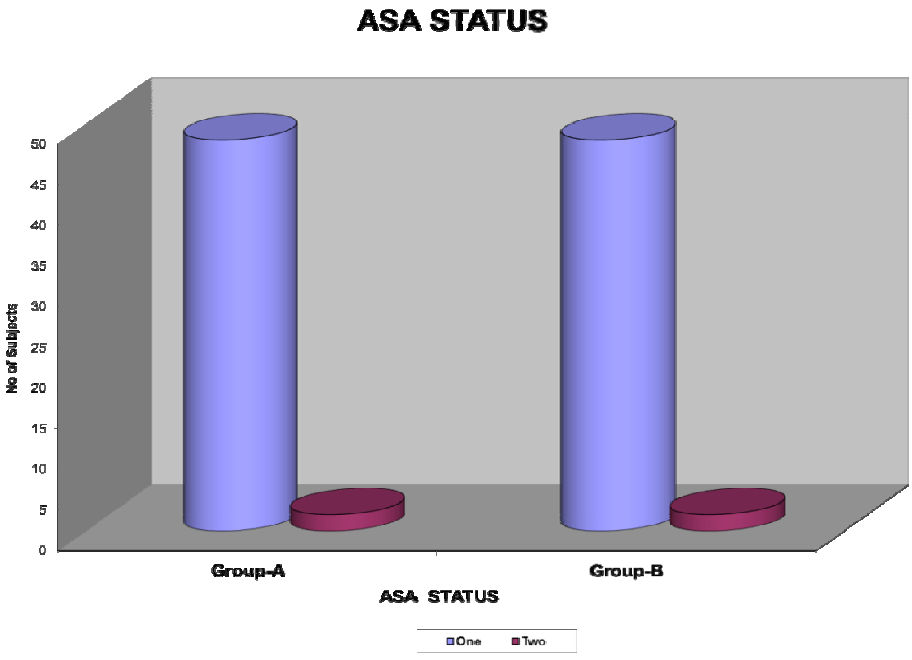


Table-5

DURATION OF SURGERY:

There was no difference in the duration of surgery between the two groups with a maximum duration of 50 min. The average duration of surgery was around 32 min in both groups.

	Group-A	**Group-B**
Mean	31.9	32.4
Sd	11.95	13.02
t-Value	**0.2**	
Df	**98**	
p-value	**0.84 (Not Significant)**	

TYPE OF SURGERY:

Type of surgery between two groups were similar .The level of blockade required was similar in both groups.

Surgery	**Group A**		**Group B**		**Total**	
	N	**%**	**N**	**%**	**N**	**%**
PVSL	2	4	3	6	5	5
URETHROPLASTY	6	12	6	12	12	12
HERNIOTOMY	16	32	13	26	29	29
PVSL+CIRCUMCISION	7	14	7	14	14	14
CIRCUMCISION	3	6	2	4	5	5
ORCHIDOPEXY	13	26	14	28	27	27
Others	3	6	5	10	8	8
TOTAL	50	100	50	100	100	100

Chi-square 1.78 DF=6 significant value =0.94 (Not Significant)

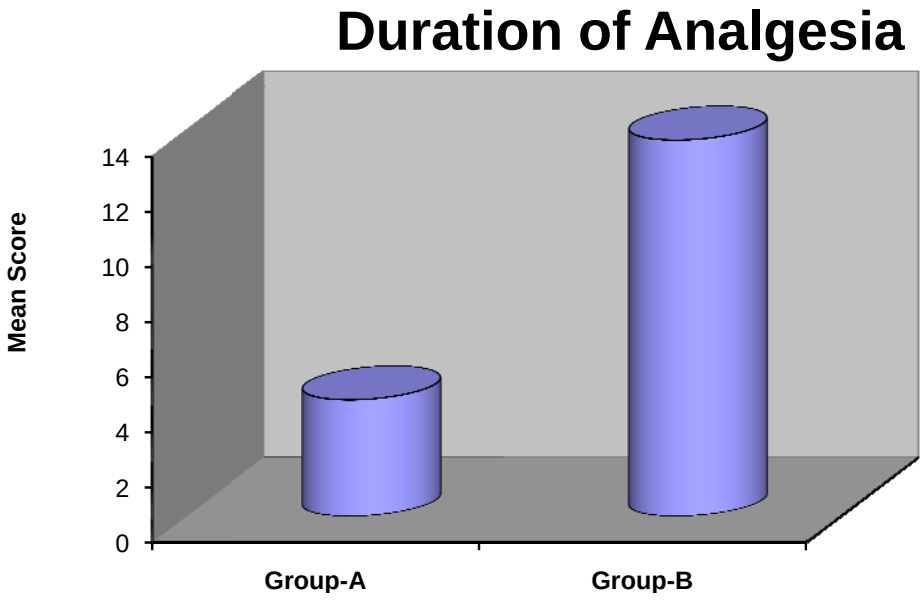
DURATION OF ANALGESIA:

There was a significant difference in duration of analgesia between two groups. Group A has post operative analgesia of (4.22+/_1.09) hours and group B has average (13.64+/_4.12) hours analgesia. Difference was statistically significant(p=0.0002)

Table-6

Duration of Analgesia

	Group-A	Group-B
Mean	4.22	13.64
Sd	1.09	4.12
t-Value	15.64	
Df	98	
p-value	0.0001 (Significant)	



Oxygen saturation:

There was no desaturation either in the intraoperative or postoperative periods in both the groups.

HEMODYNAMIC CHANGES:

We observed for change in heart rate throughout surgery and 24 hours post operatively. At the intervals of 10 minutes during first hour then 15 minutes once for two hours then half hourly and second hourly for 12 hours.. There was decrease in heart rate after caudal block in both the groups but magnitude being more in group B and was significant for first 50 minutes. The decrease in heart rate was less than 20% and the rate was maintained average around 115 none of them requiring intervention. The mean value was between 112 and 124.

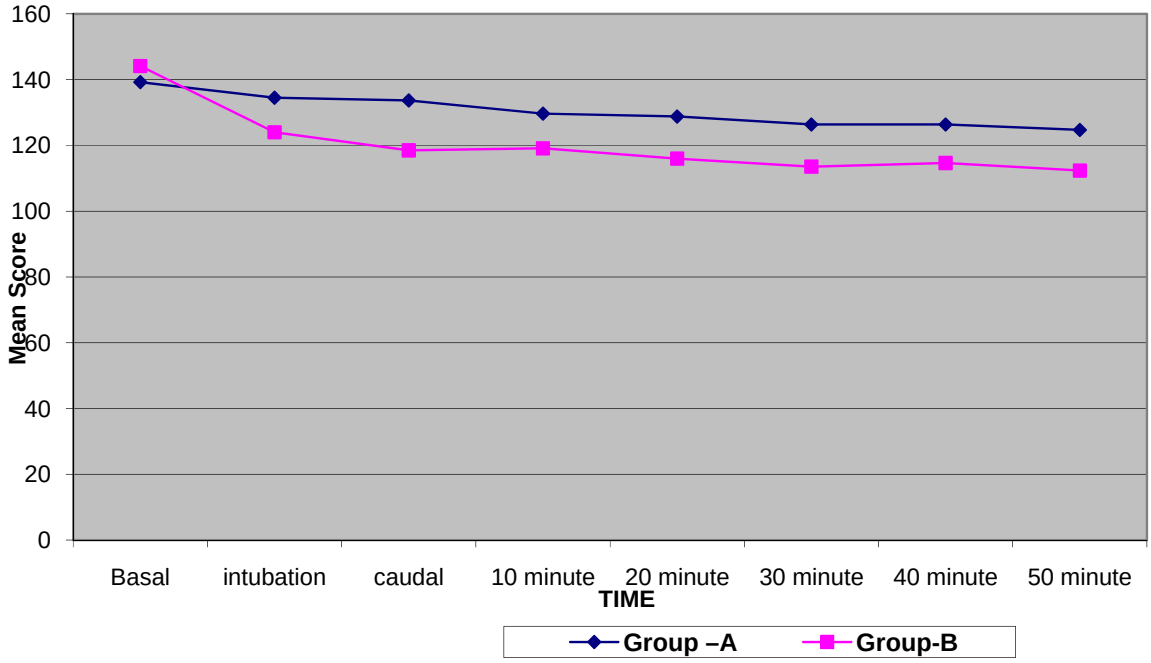
Table : 7

Heart Rate intra operative

Heart Rate	Group –A Mean ± Sd	Group-B Mean ± Sd	t-value	p-Value Df=98
basal heart rate	139.22± 17.70	144.12± 11.31	1.65	0.102
Intubation	134.48± 17.07	124.00± 10.44	3.7	0.0001*
Caudal	133.66± 16.56	118.50± 10.38	5.49	0.0001*
10 min	129.66± 14.78	119.12± 08.67	2.42	0.02*
20 min	128.76± 16.09	115.98± 09.70	4.81	0.0001*
30 min	126.36± 15.66	113.56± 17.30	3.88	0.0001*
40 min	126.34± 15.30	114.66± 09.67	0.45	0.65
50 min	124.70± 15.48	112.36± 16.06	3.91	0.0001*

* - Significant

INTRA OPERATIVE HEART RATE



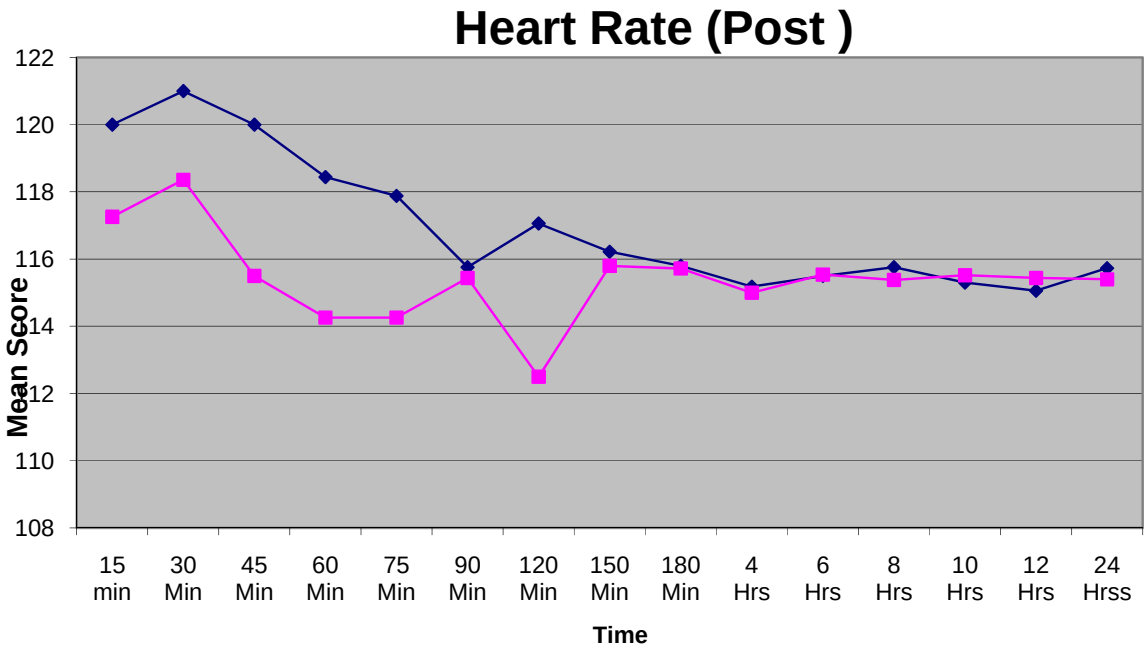
POST OPERATIVE HEART RATE: there was no significant change in heart rate between two groups post operatively. There was a mild rise in heart rate at 120 min in group A, which could be wearing up of caudal and patients starting to get pain.

Table-8

Heart Rate post operative

Heart Rate	Group –A Mean ± Sd	Group-B Mean ± Sd	t-value	p-Value Df=98
15 Minutes	120.00 ± 14.92	117.26 ± 05.83	1.21	0.23
30 Minutes	121.00 ± 12.80	118.36 ± 04.56	1.71	0.09
45 Minutes	120.00 ± 16.02	115.50 ± 07.43	1.8	0.08
60 Minutes	118.44 ± 14.02	114.26 ± 07.94	1.75	0.08
75 Minutes	117.88 ± 12.36	114.26 ± 08.08	1.73	0.09
90 Minutes	115.76 ± 20.77	115.44 ± 11.04	0.1	0.92
120 Minutes	117.06 ± 13.04	112.50 ± 17.24	1.49	0.14
150 Minutes	116.22 ± 12.96	115.80 ± 8.99	0.19	0.85
180 Minutes	115.80 ± 11.95	115.72 ± 8.92	0.04	0.97
4 Hours	115.18 ± 13.50	115.00 ± 9.59	0.08	0.94
6 Hours	115.50 ± 11.67	115.54 ± 9.01	0.02	0.99
8 Hours	115.76 ± 20.77	115.38 ± 11.04	0.1	0.92
10 Hours	115.30 ± 12.02	115.52 ± 08.99	0.11	0.91
12 Hours	115.06 ± 12.05	115.44 ± 11.04	0.16	0.87
24 Hours	115.73 ± 20.78	115.40 ± 11.06	0.07	0.92

* - Significant


Heart Rate (Post)
122
120
118
116
114
112
110
108
Mean Score
15 min
30 Min
45 Min
60 Min
75 Min
90 Min
120 Min
150 Min
180 Min
4 Hrs
6 Hrs
8 Hrs
10 Hrs
12 Hrs
24 Hrss
Time

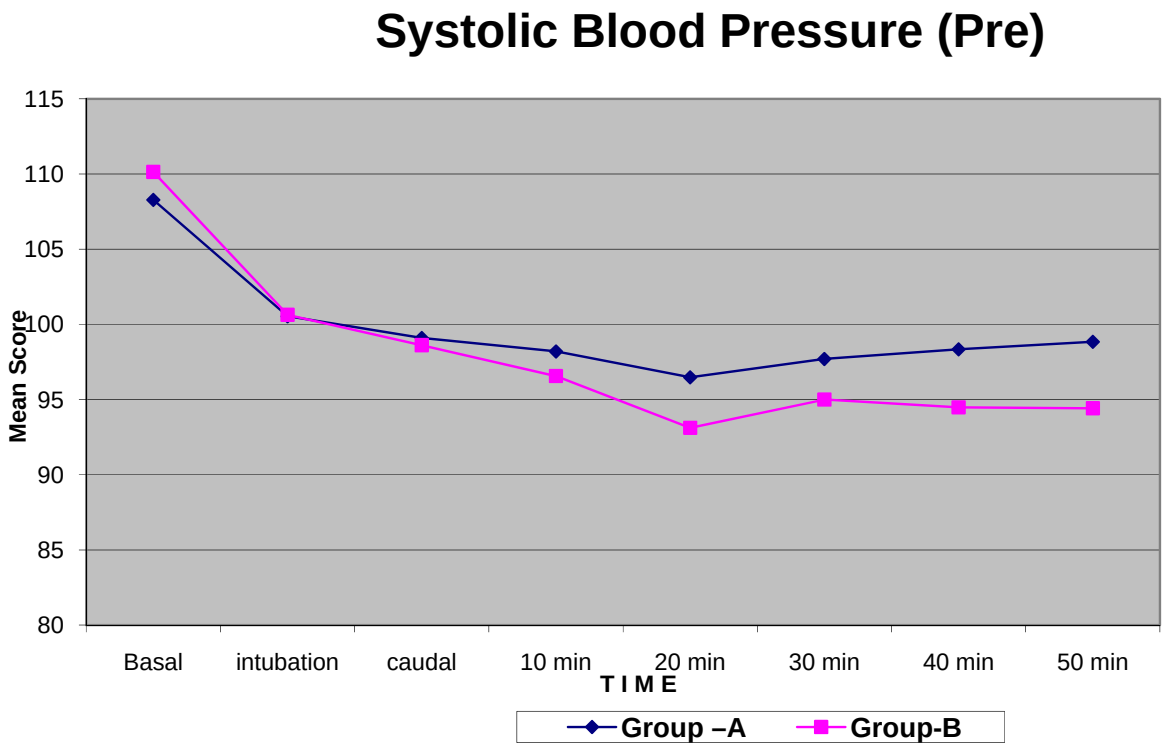
BLOOD PRESSURE CHANGE:

Pre operative basal systolic blood pressure was comparable between two groups. The mean systolic BP in group A was between 96+/-14.8 to 108+/- 8.25 .in group B mean was 94+/- 6 to 100+/- 8 intra operatively.The fall in BP was significantly more in group B compared to group A during 40 and 50 minutes after caudal block , this could be due to the absorption of dexmedetomidine and peak action on cardiovascular system. The drop in systolic BP was not > 20% which may need intervention

Systolic Blood Pressure intra operative

Systolic blood pressure	Group –A Mean ± Sd	Group-B Mean ± Sd	t-value	p-Value Df=98
Pre OP basal	108.28 ±8.25	110.14 ± 8.90	1.08	0.28
Intubation	100.54 ± 7.96	100.64 ± 8.39	0.06	0.95
Caudal	99.10 ± 8.00	98.62 ± 8.29	0.3	0.77
10 minute	98.20 ± 8.32	96.56 ±7.58	1.03	0.31
20 minute	96.48 ± 14.80	93.12 ± 15.53	1.11	0.27
30 minute	97.70 ± 8.04	95.00 ± 7.55	1.73	0.25
40 minute	98.34 ± 8.12	94.48 ± 7.35	2.49	0.01*
60 Minute	98.84 ± 8.92	94.42 ± 7.08	2.74	0.01*

* - Significant


Systolic Blood Pressure (Pre)
115
110
105
100
95
90
85
80
Mean Score
Basal
intubation
caudal
10 min
20 min
30 min
40 min
50 min
T I M E
Group –A
Group-B

POST OPERATIVE SYSTOLIC BLOOD PRESSURE:

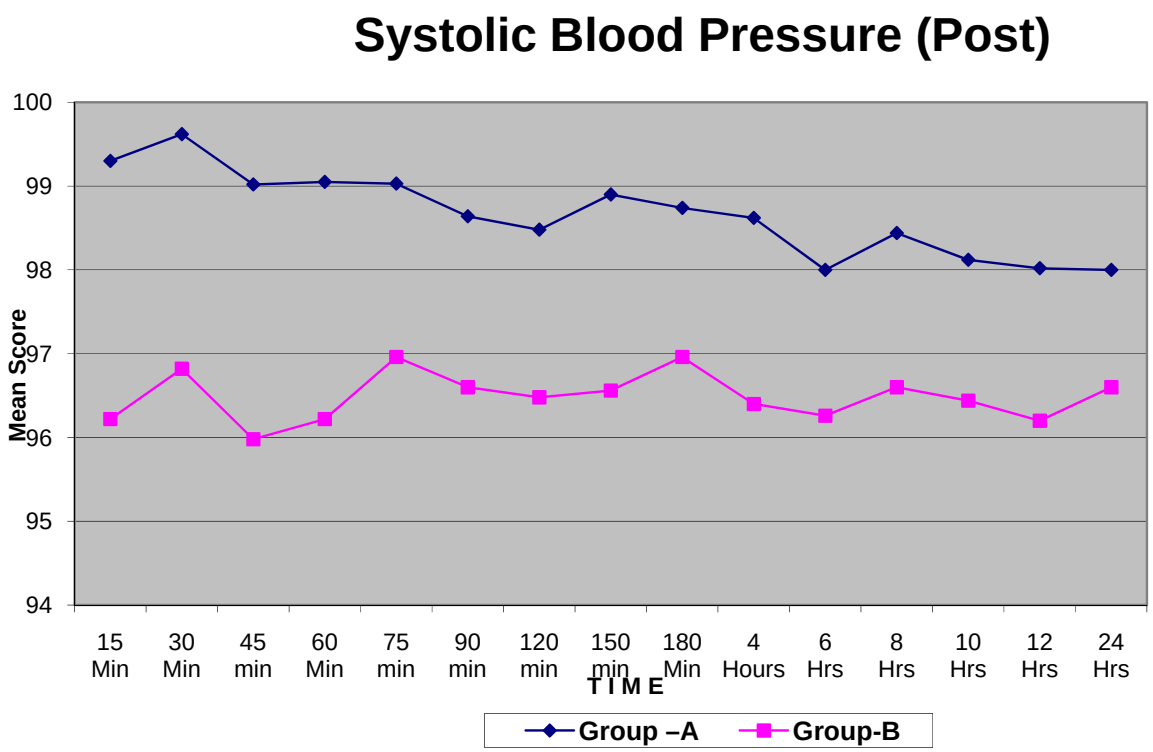
There was no significant difference between group A and group B in post operative systolic blood pressure.

Table -9

	Group –A Mean ± Sd	Group-B Mean ± Sd	t-value	p-Value Df=98
15 Minutes	99.30 ± 8.44	96.22± 9.15	1.75	0.08
30 Minutes	99.62 ± 9.72	96.82± 8.54	1.53	0.13
45 Minutes	99.02 ± 8.68	95.98± 6.94	1.93	0.06
60 Minutes	99.05 ± 8.68	96.22± 9.15	1.51	0.14
75 Minutes	99.03 ± 8.67	96.96± 7.15	1.3	0.2
90 Minutes	98.64 ± 8.55	96.60± 7.05	1.71	0.09
120 Minutes	98.48 ± 8.55	96.48± 6.59	1.2	0.23
150 Minutes	98.90 ± 7.75	96.56± 6.65	0.8	0.42
180 Minutes	98.74 ± 9.21	96.96± 7.15	1.08	0.28
4 Hours	98.62 ± 9.10	96.40± 7.09	1.36	0.18
6 Hours	98.00 ± 8.33	96.26± 6.84	1.14	0.25
8 Hours	98.44 ± 8.96	96.60± 7.05	1.19	0.26
10 Hours	98.12 ± 8.29	96.44± 6.06	1.16	0.25
12 Hours	98.02 ± 7.93	96.20± 5.60	1.33	0.19
24 Hours	98.00 ± 7.72	96.60± 6.42	0.99	0.33

* - Significant

Systolic Blood Pressure **post operative**



DIASTOLIC PRESSURE: There was slight decrease in diastolic blood pressure from base line in both the groups .there was no significant difference between group A and group B, both intra operative and post operative period.

Diastolic Blood Pressure intra operative

Table :10

Time	Group –A Mean ± Sd	Group-B Mean ± Sd	t-value	p-Value Df=98
Basal	56.46 ± 5.64	57.24 ± 6.69	0.63	0.53
Intubation	54.82 ± 5.10	55.00 ± 6.04	0.16	0.87
Caudal	54.42 ± 5.36	53.98 ± 5.61	0.4	0.69
10 minute	54.12 ± 5.09	53.76 ± 5.35	0.35	0.73
20 minute	53.74 ± 5.07	53.30 ± 5.26	0.43	0.67
30 minute	53.80 ± 5.65	53.36 ± 5.70	0.39	0.7
40 minute	53.52 ± 5.12	53.18 ± 5.03	0.34	0.74
50 minute	53.72 ± 5.25	51.86 ± 7.63	0.7	0.49

* - Significant

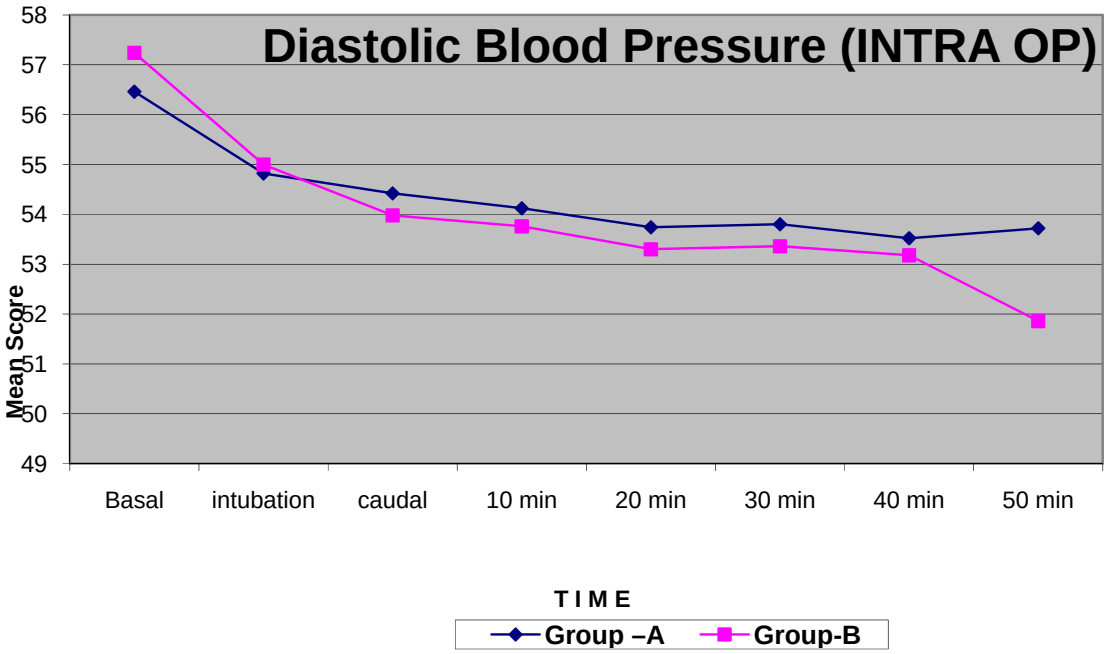
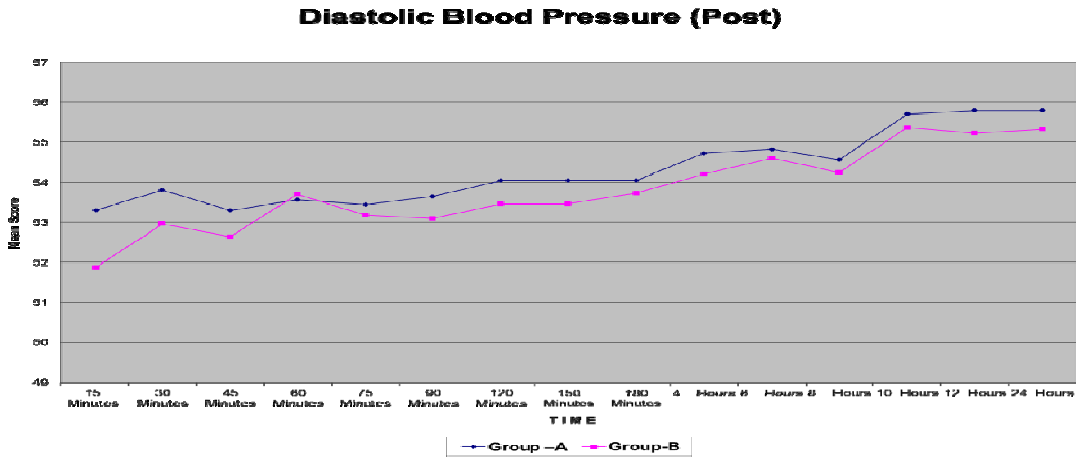

Diastolic Blood Pressure (INTRA OP)
Mean Score
58
57
56
55
54
53
52
51
50
49
Basal
intubation
caudal
10 min
20 min
30 min
40 min
50 min
T I M E
Group –A
Group-B

Table-11

Diastolic Blood Pressure **post operative**

Time	Group –A Mean ± Sd	Group-B Mean ± Sd	t-value	p-Value Df=98
15 Minutes	53.30± 4.92	51.86 ± 7.63	1.12	0.27
30 Minutes	53.80± 5.12	52.96 ± 4.59	0.86	0.39
45 Minutes	53.30± 4.68	52.64 ± 4.11	0.75	0.46
60 Minutes	53.56± 5.26	53.70 ± 4.65	0.87	0.39
75 Minutes	53.44± 5.12	53.18 ± 4.44	0.27	0.79
90 Minutes	53.64± 5.41	53.10 ± 4.93	0.52	0.6
120 Minutes	54.04± 5.36	53.46 ± 4.86	0.57	0.57
150 Minutes	54.04± 5.36	53.46 ± 4.86	0.37	0.71
180 Minutes	54.04± 5.23	53.72 ± 5.08	0.31	0.76
4 Hours	54.72± 5.48	54.20 ± 5.04	0.49	0.62
6 Hours	54.82± 5.03	54.60 ± 4.82	0.22	0.82
8 Hours	54.56± 4.44	54.24 ± 4.09	0.38	0.71
10 Hours	55.70± 4.28	55.36 ± 3.82	0.42	0.68
12 Hours	55.78± 4.60	55.22 ± 4.01	0.65	0.52
24 Hours	55.78± 4.61	55.32 ± 4.12	0.53	0.6

* - Significant

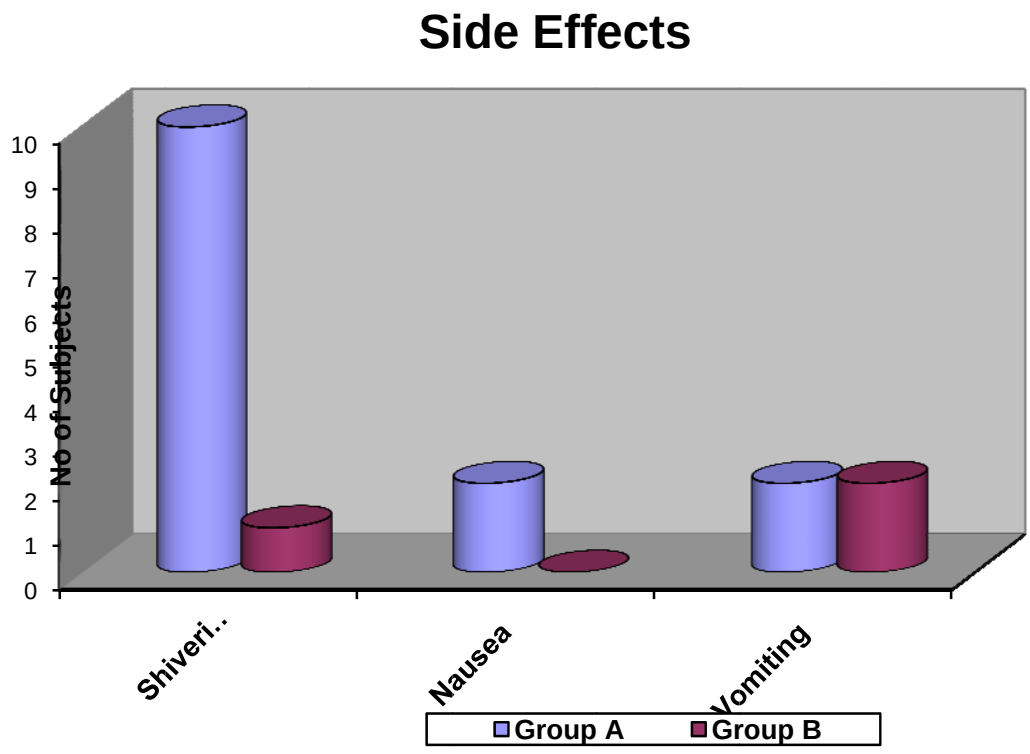


INCIDENCE OF SIDE EFFECTS:

Incidence of bradycardia, hypotension, and nausea, vomiting and shivering were compared between two groups. shivering was more common in group A (12 cases) Table-16

Side Effects

Side Effects	Group-A		Group-B	
	N	%	N	%
Bradycardia	0	0	O	0
Hypotension	0	0	0	0
Shivering	10	20	1	2
Nausea	2	4	1	6
Vomiting	2	4	2	2
Total	**14**	**28**	**4**	**8**



FLACC SCORING post operatively:

At 2 hours after surgery FLACC score was less than 3 for all the patients, none of them required rescue analgesia.

Table-12

FLACC 2 hours

FLACC Score	**Group-A**		**Group-B**	
	N	**%**	**N**	**%**
0	33	66	39	78
1	8	16	8	16
2	5	10	2	4
3	4	8	1	2
Total	**50**	**100**	**50**	**100**

FLACC score at 4 hours:

At 4 hours after caudal drug administration. 24 (48%) patients out of 50 in group A had FLACC score more than or equal to 4 . They received rescue analgesia inj fentanyl 1mcg/kg iv.

Where as in group B none of the children complained of pain and the FLACC score was less than 4 at the end of 4 hours. 4 hours

Table-12

FLACC score 4 hours	**Group-A**		**Group-B**	
	N	**%**	**N**	**%**
0	6	12	15	30
1	6	12	20	40
2	9	18	14	28
3	5	10	1	2
4	10	20	0	0
5	6	12	0	0
6	6	12	0	0
7	1	2	0	0
8	1	2	0	0
Total	**50**	**100**	**50**	**100**

FLACC score at 6 hours.

FLACC at the end of six hours only 7 (14%) children out of 50, required rescue analgesia and the pain scores were less than 5. Whereas in group A 26(52%) out of 50 children had FLACC score more than 4. At the end of 6 hours all patients in group A had received rescue analgesia.

6 hours

Table :13

FLACCscore 6 hours	**Group-A**		**Group-B**	
	N	**%**	**N**	**%**
0	0	0	2	4
1	0	0	14	28
2	6	12	22	44
3	18	36	5	10
4	8	16	5	10
5	11	22	2	4
6	3	6	0	0
7	3	6	0	0
8	1	2	0	0
Total	**50**	**100**	**50**	**100**

FLACC at 8 hours:

In group B only 4 (8%) children out of 43 had FLACC score of 4 and received rescue analgesia, remaining 46 children were calm and did not have pain. They were calm and comfortable. In group A patients who had received rescue analgesia at early (<4 hours), started complaining of pain and required extra analgesic supplementation, paracetmol 15mg/kg.

Table-14

FLACC score 8 hours	**Group-A**		**Group-B**	
	N	**%**	**N**	**%**
1	0	0	3	6
2	9	18	29	58
3	25	50	14	28
4	3	6	4	8
5	4	8	0	0
6	3	6	0	0
7	4	8	0	0
8	2	4	0	0
Total	**50**	**100**	**50**	**100**

FLACC at 12 hours:

In group B, 6 out of 31 patients required rescue analgesia. In group A 40 patients required a supplementation of analgesia.

Table-15

FLACC score 12 hours	**Group-A**		**Group-B**	
	N	**%**	**N**	**%**
1	0	0	1	2
2	2	4	11	22
3	8	16	31	62
4	11	22	1	2
5	13	26	2	4
6	9	18	1	2
7	6	12	1	2
8	1	2	1	2
Total	**50**	**100**	**50**	**100**

FLACC at 16 hours:

At 16 hours following caudal: 27 patients belonging to group B needed rescue analgesia.

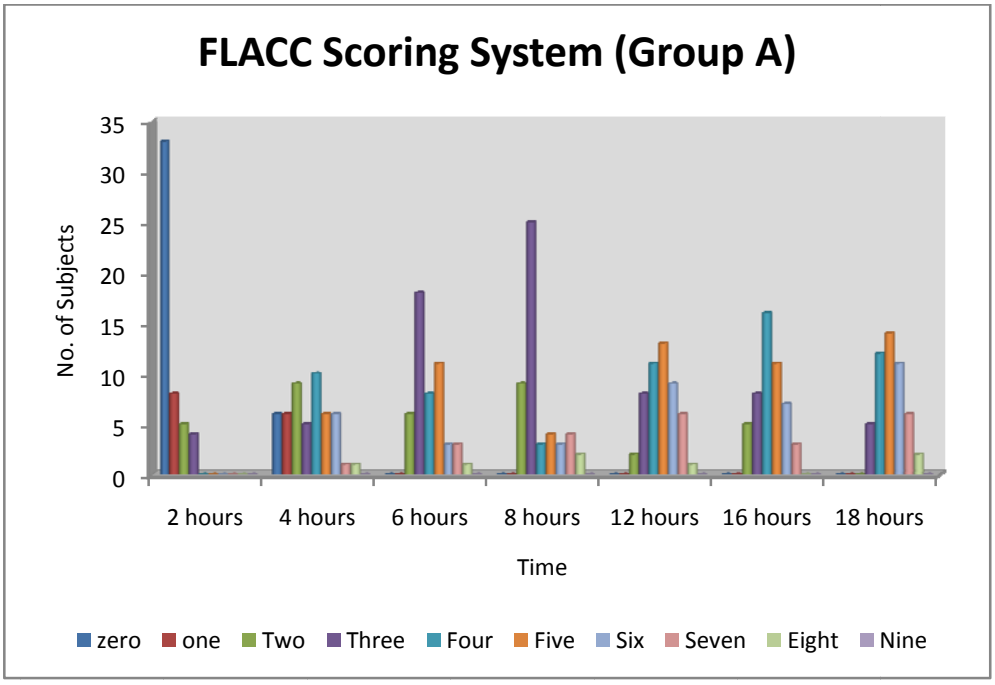
Table-16

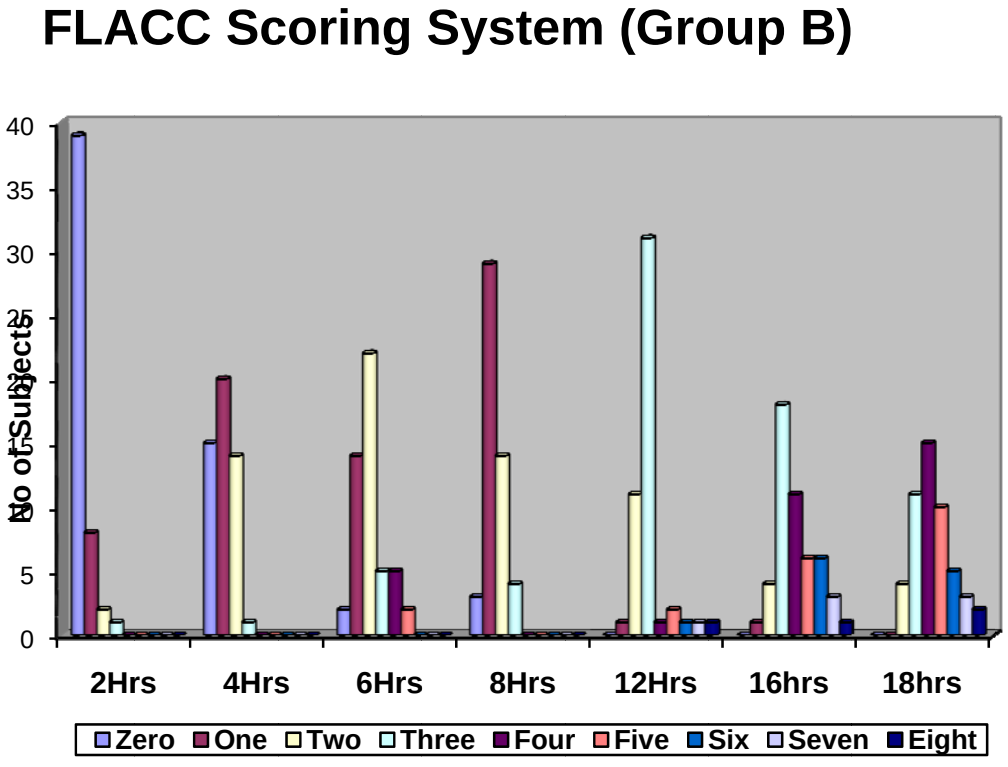
FLACC score 16 hours	**Group-A**		**Group-B**	
	N	**%**	**N**	**%**
1	0	0	1	02
2	5	10	4	08
3	8	16	18	36
4	16	32	11	22
5	11	22	6	12
6	7	14	6	12
7	3	06	3	6
8	0	0	1	2
Total	**50**	**100**	**50**	**100**

FLACC score at 18 hours. By the end of 18 hours all the patients in group B had received rescue analgesia the pain score in group B was lower compared to group A.

Table-17

FLACC score 18 hours	**Group-A**		**Group-B**	
	N	**%**	**N**	**%**
2	0	0	4	8
3	5	10	11	22
4	12	24	15	30
,5	14	28	10	20
6	11	22	5	10
7	6	12	3	6
8	2	4	2	4
Total	**50**	**100**	**50**	**100**


FLACC Scoring System (Group A)
No. of Subjects
35
30
25
20
15
10
5
0
2 hours
4 hours
6 hours
8 hours
12 hours
16 hours
18 hours
Time
zero
one
Two
Three
Four
Five
Six
Seven
Eight
Nine

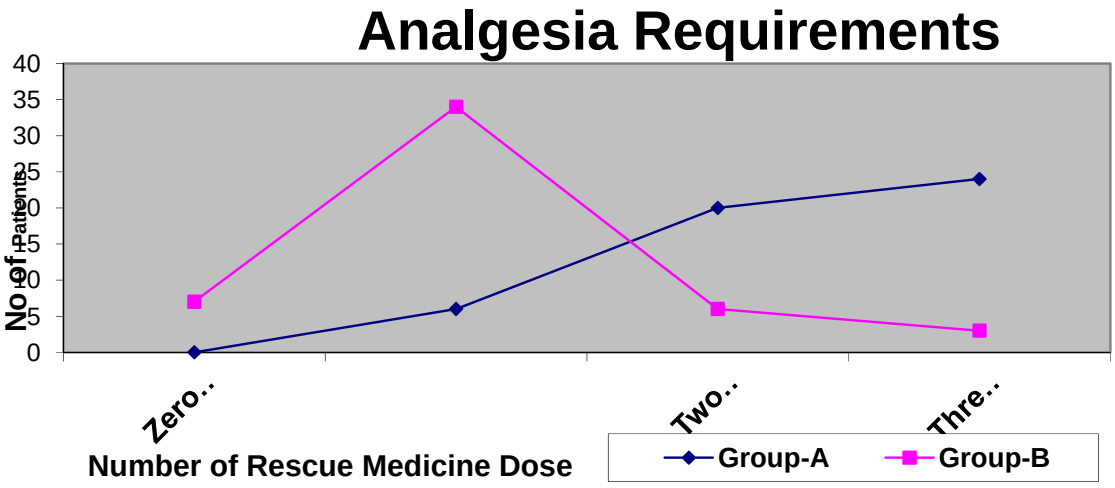

FLACC Scoring System (Group B)
No of Subjects
40
35
30
5
0
2Hrs
4Hrs
6Hrs
8Hrs
12Hrs
16hrs
18hrs
Zero
One
Two
Three
Four
Five
Six
Seven
Eight

POST OPERATIVE ANALGESIC REQUIREMENT:

The number of times patients received analgesia was more in group A compare to group B. 68% patients in group B required only once supplementation of paracetamol. 48% patients in group A received paracetmol 15mg/kg analgesia three times in 24 hour compare to only 3(6%) patients in group B.7(14%) patients in group B did not required paracetamol supplementation in 24 hours versus all patients in group A required analgesic supplementation . Number of times the additional analgesia required was more in group A and hence the total analgesia consumed is more in group A.

Post operative analgesia requirement

No of times Analgesia given	**Group-A**		**Group-B**	
	N	**%**	**N**	**%**
0	0	0	7	14
1	6	12	34	68
2	20	40	6	12
3	24	48	3	6
Total	**50**	**100**	**50**	**100**



DISCUSSION:

Eventhough neonates and children are able to perceive pain as adults do, they have been undertreated or often neglected**.** Misconceptions leading to **inadequate pain treatment** in children are: myth that children do not feel pain or remember such events, difficulty in assessing pain, deficiency in knowledge in physician about drugs strategy and newer modes of pain relief in children, fear of overdosage of pain medications and its side effects. Patient factors like improper expression of pain by paediatric patients due to lack of such experience, difference in cognitive and emotional development towards pain in children.[1]

Inadequate pain relief in paediatric age group shown to have **long term adverse effects** like disrupted sleeping and food habits, behavioural changes, harmful neuronal and endocrine responses. They are prone to perceive more pain on subsequent pain exposure. Hence pain management becomes most important component of peadiatric anaesthesia practice. [4,5,6(**anand KJ et al, peter JW et al)**

There are **different modes of pain management**[58] in post operative period for children undergoing abdominal surgeries. Oral acetaminophen , morphine which cannot be administered in immediate post operative period. Rectal administration of drugs like diclofenac , acetaminophen suppositories

have variable bioavailability and inadequate efficacy in alleviating pain .[58] Intramuscular route- painful on injection, patient compliance is poor and hence strongly discouraged

Intravenous non -opioids like paracetamol, ketorolac and opioids like morphine, pethidine, fentanyl are widely used intra operatively. Their use in post operative period is limited due to shorter duration of action needing frequent dosing intervals . Continuous infusion of opioids using PCA provide pain relief at acceptable doses and improves sleep. Opioids have increased risk of nausea , sedation[58] other drawbacks is cost effectiveness. It is difficult tp educate the child regarding use of PCA[58]. Now the nurse controlled analgesia and parent controlled analgesia are prevalent. Wound infiltration and topical anaesthetic application have limited use due to short duration of action and does not relieve visceral pain.

Diclofenac is an analgesic an weekly antipyretic NSAID available for oral. Rectal. Intramuscular and recently intravenous administration. Its analgesic effct is useful in treating acute pain in paediatrics has been proved. Oral diclofenac 0.5mg/kg is comparable to analgesic efficacy of 15mg/kg paracetamol (Standing

JF et al 2009)[60] . It has usual side effects of NSAIDS like nausea vomiting, prolonged bleeding time.

Ketorolac is a moderately anti inflammatory drug and potent analgesic when administered IM or IV. It exhibits analgesic effect by inhibiting cox enzyme and prostaglandin synthesis. It has no cardiovascular or ventilator depression action. Ketorolac can prolong bleeding time on single iv injection in spinal anaesthesia.(Thwaites et al 1996)[36]. It Can cause severe bronchospasm in patients with asthma and aspirin sensitivity.(Haddow et al)[36]. Due to side effects like GI irritation, bleed, nausea and peripheral oedema NSAIDs are not regularly used in children.

Acetaminophen is effective in mild to moderate pain management. . It has got excellent safety profile and has lesser side effects at therapeutic doses(15-20mg/kg). It acts by inhibiting central prostaglandin synthesis. The dose required to produce analgesia is more compared to requirement of drug to decrease temperature[58]. Its metabolite N-Acetyl –P –benzoquinone-imine s hepatotoxic. But growing liver produces higher level of glutathione peroxidase and is protective against hepatotoxicity in children. At higher doses P-Aminophenol accumulation causes papillary necrosis and and analgesic induced nephropathy.Acetaminophen can be administered oral rectal, intramuscular or intravenous routes. Maximum

allowable dose is 90mg[36,58,59].

Regional techniques are central neuraxial blocks and peripheral blocks. Ilio inguinal, iliohypogastric block for herniotomy, orchidopexy. Penile block for circumcision. Central neuraxial blocks like spinal, Single shot caudal epidural, continuous epidural infusion, paravertebral blocks for abdominal, perineal and lower limb orthopaedic surgeries can be used for abdominal and lower limb surgeries. **Raafat S. Hannallah,M.D., et al** (Anesthesiology 1987)[35] evaluated 44 children aged 1.5-12 years scheduled for ambulatory orchidopexy under caudal analgesia and ilioinguinal /iliohypogastric nerve blocks for postoperative analgesia. Found that caudal block is superior to ilioinguinal block

Among all above mentioned modalities of post operative pain relief **caudal block** is most commonly used since decades. It is well tolerated and most reliable method. Caudal block provides effective and extended post operative pain relief with lesser side effects. It has advantages like : local anesthetic with additives can be administered as a single injection to prolong post operative analgesia. Requires lesser number of pricks compare to regional blocks.

Caudal analgesia has shown better outcomes like early extubation, reduced length of hospital stay, early return of intestinal function[61] .Decreased requirement of anesthetic drugs and volatile intra operatively[8].

Bupivacaine is a long-acting amide local anaesthetic that has provided reliable anaesthesia and analgesia with differential motor-sensory blockade for more than 40 years.[9,10] **(steel GC et al, watt MJ et al)**. The average duration of action of bupivacaine is about 4 -6 hours. It acts by inhibiting sodium channels in the nerve membrane. Available as 0.25%,0.5% and 0.75%. The motor and the sensory blockade of local anesthetics depends on its minimum concentration(Cm) .Cm of motor fiber is twice that of sensory fibers.. 0.25% bupivacaine produces adequate motor and sensory blockade in lower abdominal surgeries. The bupivacaine is available as a racemic micture of s(-) and s(+) enantiomer, while ropivacaine is the first drug to be available as a pure S-(-) enantiomer. , ropivacaine has an intermediate degree of lipid solubility compared to bupivacaine. Ropivacaine Clearance is higher than bupivacaine and elimination half-time shorter[38] and hence shorter acting compared to bupivacaineThe volume of 1ml/kg was selected based on ARMITAGE formula.

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Local anaesthetics are commonly used either alone or with additives. Commonly used **caudal additives** are epinephrine Ketamine, midazolam, tramadol, alpha 2 agonists like clonidine, dexmedetomidine and opioids like morphine, hydromorphine, fentanyl,. (BJA 2003, miller et al) . Adrenaline 5mcg/ml at 1:20000 concentration has been used with lignocaine but its efficiency is debatable with low dose bupivacaine.[61] Morphine acts on both spinal and supraspinal mu receptors. Morphine improves both quality and duration of analgesia at 0.03,0.06 and 0.1 mg/kg dose[61]. Recently extended release epidural morphine (EREM) is available. Main side effect of morphine is late onset respiratory depression. Fentanyl 1mcg/kg is used as a adjuvant to local anaesthetics. Few studies showed no added analgesia when fentanyl was added with 0.25% bupivacaine[61]. Buprenorphine, diamorphine at 4mcg/kg dose have been tried. Side effects with opioids are nausea, vomiting, delayed onset respiratory depression[35] and urinary retention **(Gustafsson et el)**.

Clonidine has been used for sedation, premedication and analgesia.[58,61.] Studies show increase in duration of post operative analgesia on adding clonidine to local anesthetic.(Aruna parameswari et al) (Klimscha et al)[61] . Dose dependent hypotension, bradycardia are less pronounced in children [61]. Few studies showed apnea, bradycardia and spo2< 80% when used in neonates (Breschan C et al).

Dexmedetomidine. is the most recent alpha-2 agonist agent approved by FDA IN 1999 for use in humans for analgesia and sedation. Dexmedetomidine differs from clonidine as it posses selective alpha-2 adrenoceptor agonism. Action at 2A receptor makes it a much more effective sedative and analgesic agent than clonidine **(*El-Hennawy et al., 2009)*** . Addition of dexmedetomidine resulted in prolonged analgesia suggesting a synergistic effct with local anesthetics in pediatric surgery ***(kanazi et al., 2006 acta anesthesia).*** Its analgesic effect is due to its action at post synaptic alpha 2A receptor situated at substantia gelatinosa of spinal cord leading to suppressed release of substance P and hyperpolarization of nerve membrane. In addition it has got a **active sedation** property, with no respiratory depression. Hence we decided to evaluate its effect in prolonging post operative analgesia when added to bupivacaine in paediatric caudal anesthesia.

In our study we compared plain bupivacaine and effect of combination of caudal bupivacaine and dexmedetomidine in paediatric age group 2 to 7 years undergoing elective lower abdominal and perineal surgeries .In our study group A received 0.25% of bupivacaine 1ml/kg and Group B received 1ml/kg of 0.25% bupivacaine and 2mcg/kg dexmedetomidine . We observed that the duration of post operative analgesia in group A was 4.22+/- 1.52 hours, where as in group B

duration of analgesia was 13.54+/-4.12 hours. The difference was statistically significant with P value 0.001, this shows prolonged pain relief on adding dexmedetomidine 2mic/kg caudally with bupivacaine 0.25%. This findimg is supported by study done by Saadway et al, in children aged 1 to 6 years. They concluded that addition of dexmedetomidine 1mcg/kg prolongs duration of postoperative analgesia (18.5 ± 2.8hrs versus 6.2±2.8hrs)[52]. Hennaway et al who studied benefit of adding 2mic/kg dexmedetomidine to 2.5mg bupivacaine in patients less than six years age and compared with same dose of local anesthetic and clonidine. They noticed post operative analgesia for dexmed as 14-18 hours[53], whereas duration of analgesia for plain bupivacainem was 4-6 hours.

In a Indian study done by Vijay G anand et al, administration of 2mic/kg of dexmedetomidine to ropivacaine was also found to have enhanced post operative analgesia duration for about 14.5 hours[54]. For plain ropivacaine analgesia duration was only 5.5 hours.

To assess the quality and duration of analgesia, Post operative pain assessment was done using FLACC scoring system in our study. .Merkel et al evaluated FLACC scoring for assessing pain in children and found that it is reliable and valid in quantifying pain in non verbal children[23]. Group A achieved significantly

higher FLACC scores compared to group B. which was significant, Other studies like mousumi neogi et al, Hennaway et al, Kannan et al have also used same scoring system to assess post operative pain and have found it reliable in assessing pain[53,54,55].

Visual analog pain (VAS) score is self reporting score it is applicable to older children who can grade pain from no pain to severe pain. OUCHER scale has photographs of child with increasing degrees of pain, it cannot be used for younger children. Behavioral pain score like CRIES which takes account of crying, oxygen saturation, increase in heart rate and blood pressure, sleeplessness. it is designed to assess pain in term neonates. CHEOPS is applicable to 1-7 year children considers cry, facial expression, verbalization, body posture the score is 4-13.COMFORT score system can be applied to all ages but has wide range of score from 0 to 40. Among all mentioned scoring system we chose FLACC scoring because it is easy to measure scores, can be done both in awake and asleep child, it has grading of pain as mild moderate and severe pain, covers non verbal child, as its only observational score not distressing to child during post operative period.

There was a significant difference in the FLACC score between group A and group. Group A achieved higher FLACC score which was significant. We checked for FLACC score second hourly for first 18 hours after caudal block . FLACC score observed at 2hours after caudal in both groups was less than 4 and none of them required rescue analgesia.

At the end of 4 hours 24/50 children in group A had scores >4 and hence received rescue analgesia inj fentanyl 1mcg/kg i.v. In group B none of the children had pain till 4 hours after caudal block.

At the end of 6 hours 28/50 children in group A had score more than 4 and received fentanyl iv. This could be due to wear up of caudal effect of bupivacaine resulting in higher pain scores. Whereas in group B only 7 patients required, rescue analgesia at the end of 6 hours.

At 8th hour only 4 cases in group B needed rescue analgesia out of 43. Remaining39 patients were free of pain.

At 12th hour 6 out of 39 had pain and 33 children had lower PLACC score.

At the end of 14th hour 27 patients had received rescue analgesia indicating that the dexmedetomidine group had maximum of 14 hours analgesia compared to 6 hours of analgesia with plain bupivacaine.

Total number of times analgesia required in 24 hours was estimated by noting how many times each child was administered paracetamol 15mg/kg. In group A 48% children required 3 times analgesic supplementation compared to only 6% in group B. Whereas 82% of children in group B required analgesia for only once in first 24 hours after surgery. And hence dexmedetomidine addition to caudal bupivacaine significantly reduces post operative analgesic consumption and better pain relief in children. This is supported by Hennaway et al, Kannan et al and mousumi neogi et al and others[53,55,57].

During observations in our study we found that the basal hemodynamic parameters like heart rate , systolic and diastolic BP were comparable in both groups There was fall in mean heart rate in both the groups after caudal block. The fall in heart rate was more in group B than group A and it was statistically significant. The reduced heart rate was not > 20% from basal heart rate and hence none of the children required treatment for decreased rate. The fall could be due to reduced circulating catecholamines in group receiving dexmedetomidine in addition to sympathetic blockade by bupivacaine .The mean heart rate in group A during surgery was between 124 – 139 group B 112. Post operative heart rate was 115-121 in group A, where as it was constantly around 115 in group B without much fluctuations . There was a mild rise in mean heart rate at 120-150 minutes post operatively in group A, which corresponds to 3-4 hours after caudal block, which could be due to wearing up of caudal effect of bupivacaine and patients starting to get pain.

The mean systolic blood pressure in group A was around 98 intra-operatively and between 98 and 108 post operatively. Whereas in group B mean systolic blood pressure was around 93 intra operative and 96 in post-operative period . During 40th and 50^{th} minute after caudal block there was significant decrease in systolic blood pressure in group B but the fall was not > 20% from the basal so we did not intervene any of the patients. This fall was may be due to the systemic absorption of dexmedetomidine and its peak action on cardiovascular system. Studies show the peak action on systolic BP is around 20 minutes after i.v. administration of dexmedetomidine.

Post operative decrease in mean systolic blood pressure in Group B and group A was statistically not significant. The change in mean diastolic blood pressure between two groups was not statistically significant both intra operatively and post operatively.

. Hence we conclude from our observations that All the children in the study were hemodynamically stable during surgery and 24 hours after. None of the children had bradycardia or hypotension requiring intervention. . This is supported by Hennaway et al, vijay G et al and Saadway et al they found no clinically significant hemodynamic changes in their study[52,53,54,55,57].

Intraoperatively oxygenation was maintained with 50% oxygen with nitrous oxide.there was no fall in saturation in either group. Post operatively spo2 was above 97% in all patients in room air none of them required oxygen supplementation. And hence there was no incidence of respiratory depression in plain bupivacaine or dexmedetomidine group. This finding is similar to other studies like Hennaway et al, Saadaway et al, Mousumi neogi ET al.

Incidentally we noticed occurrence of side effects like shivering, and nausea vomiting. 20% of children in group A had shivering whereas in group B only 1 (2%) child had shivering. The lesser incidence of shivering in group B is due to dexmedetomidine (Sukhminder singh et al)[66] action on thermoregulatory centre in the hypothalamus, which sets the core temperature and increases threshold for shivering.

The incidence of nausea and vomiting was not statistically different in both groups. In group A 2 patients had nausea and one patient had vomiting which was treated with injection ondonsetron 0.06 mg/kg. In group B one child had vomiting and the one patient had nausea.

From above observations and comparisions we conclude that the addition of dexmedetomidine 2mcg/kg to caudal 0.25% bupivacaine 1ml/kg significantly increases

duration of post-operative analgesia in children of 2-7 years age undergoing elective sub umbilical surgeries. Addition of dexmedetomidine provides stable hemodynamics and lesser incidence of shivering in patients. The incidence of side effects are not increased on adding dxmedetomidine to caudal bupivacaine.

SUMMARY:

Bupivacaine is a long acting amide local anesthetic. It is most frequently used for caudal anaesthesia in children that provides effective analgesia and motor blockade.

Dexmedetomidine is a potent alpha 2 agonist, widely used to provide analgesia sedation and anxiolysis. It is a safe adjuvant to to bupivacaine in pediatric caudal anaesthesia.

In our study we evaluated the effect of combination of bupivacaine and dexmedetomidine in prolongation of post operative analgesia in children. Incidentally hemodynamic changes and side effects like nausea vomiting and shivering was also compared between plain bupivacaine group and combination of bupivacaine and dexmedetomidine group, in children undergoing lower abdominal and perineal surgeries.

In a double-blinded comparative study, 100 children aged 2-7 years of ASA I and II physical status were randomly allocated to receive a single pre-surgical caudal injection of 1ml/kg of 0.25% bupivacaine and 1ml normal saline (Group A) or 0.25% Bupivacaine and 1ml of 2mcg/kg dexmedetomidine (Group B), after induction of general anaesthesia. Apart from monitoring the vital parameters likr, heart rate, blood pressure, spo2, all children were assessed for postoperative analgesia by FLACC pain scale. Incidence of side effects like nausea vomiting and shivering was noted.

The two groups were comparable for age, sex, weight, vital signs, duration and type of surgery. The following results were noted at the end of study.

1. Thequality and duration of analgesia was significantly prolonged in group B(14+/-69 HOURS) compared to group A (4.22 hour).

2.Total number of analgesic administered after rescue analgesia in group A was very high compared to group B.

3, hemodynamic changes between two groups were not significant in both groups. There was no bradycardia or hypotension either intra operatively or post operatively.

4. Incidence of shivering was high in group A compared to group B.

From above results we conclude that dexmedetomidine is a safe and effective adjuvant to local anaesthetic bupivacaine for paediatric caudal anaesthesia. Dexmedetomidine 2mcg/kg with bupivacaine 0.25% 1ml/kg provided quality analgesia and extended duration of post operative analgesia compared to plain bupivacaine 0.25% in equal volumes and concentration when administered for caudal block for sub-umbilical surgeries. Dexmedetomidine provided hemodynamic stability and less incidence of shivering in the post operative period compared to plain bupivacaine.

CONCLUSION:

From above observations, our study allow us to conclude that the addition of dexmedetomidine 2mcg/kg to caudal 0.25% bupivacaine significantly increases duration of post-operative analgesia in children of 2-7 years age undergoing elective sub umbilical surgeries. Addition of dexmedetomidine provides stable hemodynamics and lesser incidence of shivering in paediatric patients.

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PROFORMA

- DATE:
  SERIALNO:
- NAME:
- GROUP A/B :
- AGE/SEX: WEIGHT: IP.NO:
- DIAGNOSIS:
- SURGERY PLANNED:
- ASA STATUS:
- ASSOCIATED MEDICAL CONDITIONS:
- PREMEDICATION:
- IV ACCESS:
- MONITORS: HR: BP: SPO2
- CAUDAL BLOCK TIME:

- VOLUME OF LOCAL ANAESTHETIC:
- INCISION TIME(ONSET TIME):

- INTRA OPERATIVE MONITORING

Time in minutes	Basal	Intubation	Caudal	10	20	30	40	50
PR								
BP								
SPO2								

POST OP MONITORING

TIME in minute	15	30	45	60	75	90	120	150	180	4	6	8	10	12	24
HR															
SBP															
DBP															
SPo2															

FLACC PAIN SCORING for post op monitoring

CATOGORIES	SCORING 0	1	2
FACE			
LEGS			
ACTIVITY			
CRY CONSOLABILITY			

- TIME OF FIRST RESCUE ANALGESIC DRUG ADMINISTRATION:FLACC score

>4: injection fentanyl 1mcg/kg i.v. To achieve score <3

- ANY ADDITIONAL ANALGESIC REQUIRED:
- **Incidence of shivering, nausea, vomiting**

TOTAL DOSE OF ANALGESIC REQUIRED

INCIDENCE OF HYPOTENSION AND OR BRADYCARDIA NEEDING INTERVENTION